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ACOUSTIC SOUND FILTRATION AND HEARING AIDS

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The purpose of this paper is to discuss the acoustic properties of the external canal of the ear and of the middle ear with special reference to the fitting of hearing aids and the design of individual ear molds.

If the receiver of a hearing aid is connected with an individual ear mold and inserted into the ear canal, it can be observed that the high frequency partial tones of the speech sound seem rather weak. This results in greater difficulty in distinguishing consonants like s, th, f and similar sounds. When the hearing aid is connected with a flat ear piece, the mentioned tones are perceived more clearly than with the first arrangement. It is also observed that patients suffering from perceptive hearing loss hear better with a flat ear piece than with a so-called midget receiver connected to an individual ear mold. The final choice is the latter arrangement, because it is less conspicuous and easier to wear.

It seems, therefore, as has been stated by Littler,[1] that the present design of the ear piece causes serious loss in the upper frequencies. Since the transmission of the high partial tones of speech is essential for the fitting of patients with perceptive hearing loss, a discussion of the acoustic line between the receiver and the drum is assumed to be justified.

It is necessary first to review the acoustic properties of the head and the external canal of the ear under normal conditions. The principal investigators of this problem were Ballantine,[2] West,[3] and Békésy.[4] If the head is placed in a free sound field, the sound pressure on the surface of the head is different from that in the undisturbed sound field. It has been computed and experimentally verified that the sound pressure at the head increases with increasing frequency up to twice the amount of pressure in the undisturbed field. There are also differences of the pressure in front of the external canal and in that at the drum. It can be seen from figure 1 *A* that the pressure at the drum is as much as three times as large in the important frequency range between 2000 and 3000 cycles per second. Figure 1 shows the difference in sound pressure at the drum with reference to that measured in the undisturbed sound field. Dickson[5] assumed that the resonance in the external canal may be responsible for the tonal dip at 4096 cycles found in audiograms of people exposed to noise. Observing audiograms (such as fig. 1 of Wever[6]), one frequently encounters a small dip at 512 cycles. This would correspond to the small

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1. Littler, T. S.: J. Scient. Instruments **13**:144, 1936.
2. Ballantine, S.: Physiol. Rev. **32**:988, 1928; Proc. Inst. Radio Engin. **18**:1216, 1930.
3. West, W.: Inst. Electric. Engin. J. **68**:441, 1930.
4. von Békésy, G.: Ann. d. Physik. **14**:51, 1932.
5. Dickson, E. D. D.: Bull. War Med. **1**:197, 1941.
6. Wever, E. G.: Laryngoscope **52**:169, 1942.

resonance peak between 500 and 1000 cycles as seen in my figure 1 and would tend to support the theory of Dickson.

The form of the resonance curve depends on the density of the medium through which the sound passes (normally air), the length of the external canal, its cross-sectional area and the impedance of the drum. Any change in the dimensions of the factors mentioned would result in a change of the curves in figure 1. Littler[7] demonstrated changes of the resonance during filling of the external canal with hydrogen. Helmholtz[8] observed a shift in the peak of resonance in performing Valsalva's experiment. This was due to a change of the impedance of the drum, as has been verified by Lucae.[9] Incidentally, the latter performed the first clinical studies on impedance of the drum, utilizing Quincke's experiment with the interference tube.[10] Lucae, knowing of the importance of resonance in the external canal to recognition of speech, complained that hearing tubes shift the peak of resonance down toward the low frequencies, explaining it on the ground of the elongation of the external canal.[11] If the canal is made shorter, the peak is shifted to the higher frequencies. An ear mold inserted into the canal actually shortens it, causing such frequency distortion. As will be seen later, there are additional factors involved in the ear mold of present design which cause serious frequency distortion.

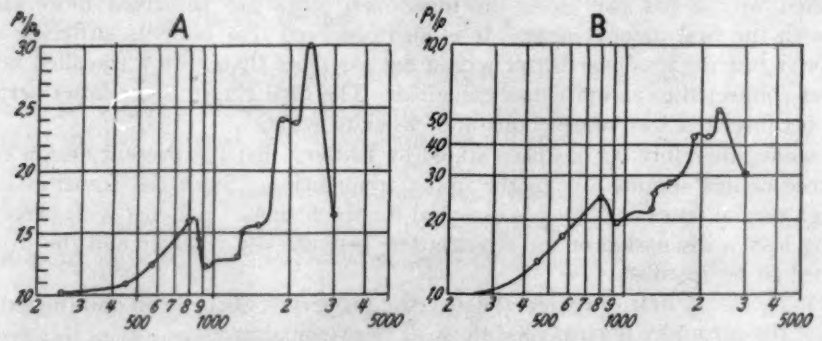

Fig. 1.—*A*, increase of sound pressure due to resonance in the external canal of the ear. p_0 represents sound pressure in front of the external canal; p_1, sound pressure at the drum. *B*, total increase of sound pressure at the drum as compared with the sound pressure in the free sound field. p represents sound pressure in the undisturbed field; p_1, sound pressure at the drum. (After von Békésy.[4])

In view of the importance of the distribution of power in speech sounds to the problem, a short consideration of speech energy is of value.

Figure 2 shows the average total speech power and its distribution with reference to frequency. The investigation was made by Dunn and White.[12] It demonstrated that 95 per cent of the total speech power is attached to the partial tones below 1000 cycles. Only 5 per cent of the total power is available for the higher partial tones, so important to the intelligibility of speech. The measurements for

7. Littler, T. S.: Nature, London **143**:118, 1939.

8. von Helmholtz, H.: Die Lehre von den Tonempfindungen als physiologische Grundlage für die Theorie der Musik, ed. 4, Braunschweig, Friedrich Vieweg und Sohn, 1877, p. 187.

9. Lucae, J. C. A.: Zur Entstehung und Behandlung der subjectiven Gehörempfindungen, Berlin, O. Enslin, 1884, p. 6.

10. Lucae, A.: Arch. f. Ohrenh. **3**:186, 1867.

11. Lucae, A.: Die chronische progressive Schwerhörigkeit, Berlin, Julius Springer, 1907, p. 337.

12. Dunn, H. K., and White, S. O.: J. Acoust. Soc. America **11**:278, 1940.

figure 2 were made at a distance of 30 cm. from the mouth. The relation of the resonance phenomena in the external canal to intelligibility becomes more evident with reference to the curve prepared by Dunn and White.[12]

EXPERIMENTS

In order to investigate the action of molds on the transmission of sound from the receiver to the drum, I had three individual ear molds prepared, each having a different diameter of the sound-conveying canal. Mold 1 is the conventional mold having a canal diameter of approximately 3 mm. Mold 2 has a diameter of 1.5 mm., and mold 3, a diameter of 5 mm. The length of each of the canals is about 22 mm. The molds were connected with the receiver of a vacuum tube hearing aid in succession. The tests were made in a normal office room. The examiner spoke into the microphone of the aid from a distance of 10 feet (300 cm.). The unaided ear was masked by moving the index finger rapidly in the external canal of the unfitted ear. The experimental conditions in testing the three molds were unchanged.

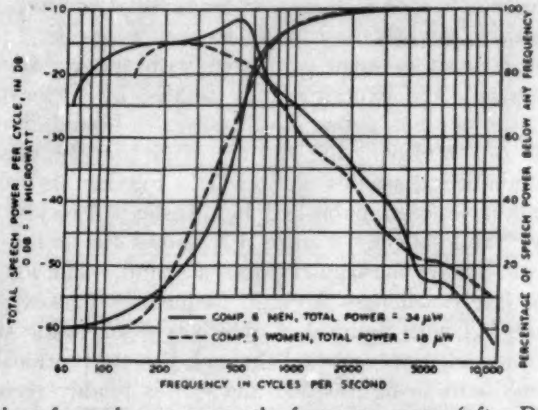
TOTAL SPEECH POWER PER CYCLE, IN DB
0 DB = 1 MICROWATT
PERCENTAGE OF SPEECH POWER BELOW ANY FREQUENCY
COMP. 6 MEN, TOTAL POWER = 34 μW
COMP. 5 WOMEN, TOTAL POWER = 18 μW
FREQUENCY IN CYCLES PER SECOND

Fig. 2.—Distribution of speech power over the frequency range (after Dunn and White [12]).

The criterion of experimentation was the brightness of speech sound and its intelligibility. The brightness of a sound impression is dependent on the amount of higher partial frequencies. The brighter impression is the one embracing the larger portion of higher partial frequencies.[13] If the same sounds are observed under different conditions of sound transmission, "brightness" and "dulness" can be interpreted as a consequence of frequency distortion. The conditions of experimentation were chosen in such a way that the hearing aid connected with mold 1 gave good intelligibility. As pointed out, the impression was that the high partial tones were weak. It was quite a strain to make out the consonants, the recognition of which depends primarily on upper partial frequencies. The results with mold 2 were rather startling. Speech sounded less loud than with mold 1, but after adjusting the volume to a comfortable loudness it was almost impossible to understand a single word. The words sounded dull, and the impression was that the higher frequencies were cut off entirely. On the other hand, when mold 3 was used, speech sounded brighter than when mold 1 was used, and more natural. It is believed that in this experiment the "naturalness" depends on the smallest inter-

13. Stevens, S. S., and Davis, H.: Hearing, New York, John Wiley & Sons, Inc., 1938, p. 165. Lichte, W. H.: J. Exper. Psychol. **28**:455, 1941.

ference of all three molds with the normal dimension of the aural canal and also on the lack of filter action in mold 3.

Another experiment was performed, that of placing a tube with a side branch orifice between the receiver and mold 3. The tube was 5 mm. long and had a diameter of 4 mm., while the side branch had a diameter of 1.2 mm. and a length of 1 mm. When the latter arrangement was used, the loudness was considerably reduced as compared with that experienced when the arrangement without the tube was used. Moreover, when the volume control had been adjusted to yield a comfortable loudness, the difference in loudness between parts of speech producing peaks of speech power and those producing small speech power was smaller than the difference in loudness when the adjustment without the little tube was used. Disagreeable loudness due to peaks of speech power is known as "banging."

COMMENT

The observations had been anticipated, since the dimensions of the external canal and the mold are ideal for acoustic sound filtration. It is known from the studies on the latter that the insertion of a channel in an acoustic conduit, either expanding or constricting in nature, renders the conduit frequency selective, provided that the cross section of the conduit is small compared with the wavelength. The basic problem was theoretically and experimentally treated by Stewart.[14] The experiments described in the present paper on filtration of sound in the acoustic line between the receiver of the hearing aid and the drum and the practical conclusions are based on the fundamental studies of Stewart. Further theoretic treatments of the general problem have been published by Mason,[15] Lewy,[16] Waetzmann and Noether,[17] Lindsay [18] and others. Figure 3 *A* shows the effect of a constricting channel in an acoustic line on the transmission of sound. The low frequencies pass through the conduit freely, whereas the high frequencies pass with more difficulty. Now compare figure 3 *A* with figure 4 *A*, which is a schematic sketch of the conditions under which sound is transmitted through the conventional ear mold. The similarity of the conditions in figures 3 *A* and 4 *A* is readily recognized. The ear mold of present design renders the acoustic line between the receiver and the drum a finite low pass filter. The longer the inserted sound canal of the mold and the smaller its diameter, the stronger is the filter action. The latter is the result of the intersection of a reactive component, an inertia, in series with an acoustic line. The viscosity of the air in the inserted channel represents a resistive component. It was a logical consequence to try a reactive component in an acoustic line in parallel. Such a component is a side branch orifice in an acoustic conduit. Figure 3 *B* shows the transmission characteristics of a tube with side branch orifices. With reference to the requirements of fitting patients with perceptive hearing loss, it is of interest to note that the high pass filter action is gradual. A cut-off frequency appears when several side branches are used along the line. Figure 4 *B* shows the special application to the ear of the general experiment the results of which are to be seen in figure 3 *B*.

Now consider some requirements for the fitting of a hearing aid to a patient suffering from perceptive hearing loss. Low frequencies have an unfavorable

14. Stewart, G. W.: Physical Rev. **20**:528, 1922. Stewart, G. W., and Lindsay, R. B.: Acoustics, New York, D. Van Nostrand Company, 1930, pp. 159-189.
15. Mason, W. P.: Bell System Tech. J. **6**:258, 1927; Physical Rev. **31**:283, 1928.
16. Lewy, E.: Physikal. Ztschr. **31**:358, 1930.
17. Waetzmann, E., and Noether: Ann. d. Physik **13**:212, 1932.
18. Lindsay, R. B.: J. Applied Physics **9**:612, 1938.

effect on the intelligibility of speech for a patient with this disability. They mask the higher frequencies considerably. Another reason is that the loudness of speech sound is due chiefly to its low frequency content. Since patients suffering from perceptive hearing impairment have a pathologic loudness reaction (the recruitment phenomenon [19]), they have a smaller band of comfortable loudness, compared with the intensity band, than persons with normal function. A loud sound which is quite tolerable for normal persons produces intolerable, even painful sensation in the ears of a patient suffering from the said disability. Loud speech sounds—and if they are loud, they must contain low partial frequencies of relatively high intensity—produce the sensation of banging, which is one of the major difficulties in the fitting of a hearing aid to a person with perceptive hearing loss. A drastic reduction of the intensity of low partial frequencies in transmission of speech has been suggested, therefore, for the fitting of persons who have perceptive hearing impairment.[20] The high frequencies, on the other hand, should be

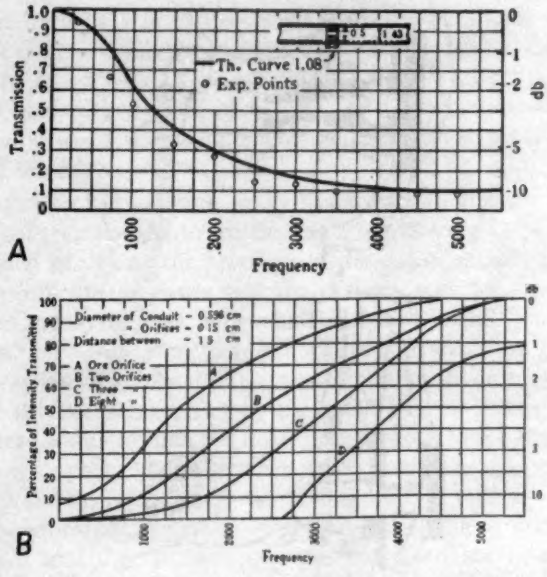

Fig. 3.—*A*, transmission of sound through a tube with a constriction (after Stewart and Lindsay [14]). *B*, transmission of sound through a tube with side branch orifices (after Stewart and Lindsay [14]). *Th. curve* stands for theoretic curve. *Exp. points* stands for experimental points.

transmitted with the highest possible fidelity. Persons with perceptive hearing loss are particularly sensitive to the nonlinear type of sound distortion. It has been a clinical observation that the sensitivity toward nonlinear distortion increases with increasing loss of perception of high frequencies.[20b]

Consider now the parts of a hearing aid used at present in the fitting to a person with perceptive hearing loss. A vacuum tube amplifier with a crystal microphone and a crystal receiver, producing a minimum of parasitic noise, is connected with an individual ear mold of current design. The amplifier has an electrical reactive circuit, an electrical high frequency pass filter of either the fixed or the variable type. (A

19. Fowler, E. P.: Tr. Am. Otol. Soc. **27**:207, 1937.

20. Halsted, T. H., and Grossman, F. M.: (*a*) New York State J. Med. **41**:352, 1941; (*b*) **42**:1944, 1942.

comprehensive description of several types is given by Littler.[21]) Why is it that there is still considerable "banging" in spite of the fact that the low frequencies have been attenuated electrically? The answer seems to be that while the electrical high pass filter attenuates the low frequencies in the amplifier, the acoustic low pass filter, as represented by the mold of present design, attenuates the high frequencies, thus neutralizing the filter action in the amplifier. The patient who needs high frequencies urgently for intelligibility turns on the volume control in order to get more of them. Unfortunately, the low frequencies are then amplified correspondingly, enough to produce the uncomfortable "banging." The design of mold for the fitting of patients who suffer from insufficient perception of high

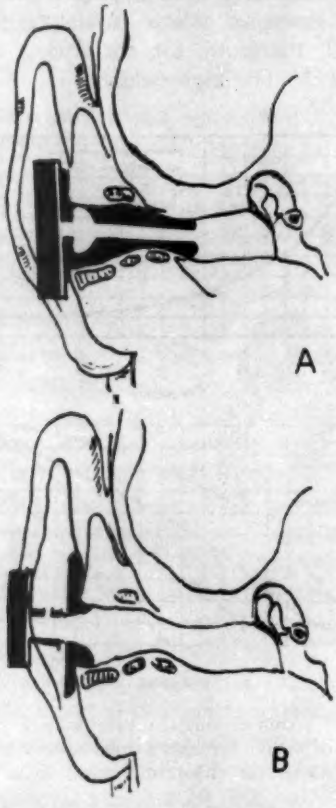

Fig. 4.—*A*, schematic sketch of a receiver connected with the ear by means of a mold of present design. *B*, schematic sketch of a receiver connected with the ear by means of a mold having a short tip and a wide sound-conveying canal. A high frequency pass filter (short tube with side branch orifice) is interposed between the receiver and the mold. This arrangement is for perceptive hearing loss.

tones must be such as to prevent attenuation of the high frequencies and such as to interfere as little as possible with the original acoustic properties of the external canal. It is suggested therefore, that the tip of the mold be short and that the sound-conveying canal should be as large and straight as possible. The use of an acoustic high pass filter is recommended in fitting for this disability.[20b] Although quantitative measurements have not been made, the action of the side branch orifice in connection with the acoustic line between the receiver and the drum must be

21. Littler, T. S.: Wireless World **46**:205, 1940.

considerable in reducing the transmission of low frequencies. This can be concluded from the reduction of the loudness produced by speech sound as compared with the loudness produced without the filter. Patients using an acoustic high pass filter with the new design of mold claim to hear more natural speech sounds, and the banging is still more reduced. Since the transmission characteristic of an acoustic conduit with side branch orifices depends partly on the length and on the cross-sectional area of the latter, a variable acoustic tone control has been developed which consists of two tubes. The outer one has a number of holes drilled in one half of its circumference. The inner tube has a slit. When the inner tube is turned in the outer one, the slit permits the holes to become side branches. If sound filtration is not desired, the inner tube is brought into such a position that it covers all the holes of the outer one. This variable acoustic tone control is placed between the receiver and the mold and is designed in such a way as to permit variation of the frequency distortion characteristic, like that produced electrically. Likewise, volume control, is obtainable to a limited extent by simply turning the receiver.

The acoustic high pass filter described is preferable to the electrical equivalent used in some hearing aids at the present time because (1) the number of parts in the amplifier can be reduced, thus simplifying it and giving a chance for further reduction of its size without loss of quality, (2) because of simple operation and (3) because, owing to its very simple construction, the acoustic tone control is less liable to get out of order.

It is an old practice in fitting a patient who had middle ear hearing impairment with a hearing aid to make the tip of the ear mold as long as possible. This design has been advocated lately and the principle of the use generalized by Schier.[22] With respect to this problem, the drum and the ossicles may be considered as a simple mechanical vibrating system as suggested by Mach,[23] the limitations imposed by such a simplification being kept in mind. It was Itard,[24] by the way, who made mention of the earliest conception of the ossicles as an involuntary vibrating system, perceived in the seventeenth century by the great French physician J. G. Duverney. In taking a typical audiogram of the impairment of hearing caused by impedance in the middle ear it appears that the lower the frequency the greater is the force necessary to provoke threshold excitation. One may as well say that the mechanical impedance of the vibrating system is increased as compared with the system under normal conditions and that it is chiefly the increased stiffness of the impedance which is responsible for the increased force of low frequency necessary for appropriate displacement. A comprehensive graph showing the influence of mass and stiffness on a vibrating system is given by Lowy.[25] Fowler [26] is of the opinion that most pathologic changes in the middle ear influence the stiffness ("elasticity") factor of the vibrating system, increasing it. In order to overcome the increased impedance of the pathologically changed mechanism of the middle ear, better transmission of power is required from the receiver to the drum. This is accomplished by an increase of the stiffness of the air in the channel between the receiver and the drum. An increase of stiffness of an air space can be accomplished by reducing its volume. This is effectively done by making the tip of the ear mold as long as possible, so that it replaces air on being inserted into the external canal. The sound-

22. Schier, M. B. A.: Laryngoscope **51**:52, 1941.
23. Mach, E.: Sitzungsb. d. k. Akad. d. Wissensch. Math-naturw. Kl., Oct. 8, 1863, p. 283.
24. Itard, I. M. G.: Maladies de l'orielle, ed. 2, Paris, Méguignon-Marris fils, 1842, vol. 1, p. 80.
25. Lowy, K.: J. Acoust. Soc. America **13**:383, 1942.
26. Fowler, E. P.: Theoretical Interpretations of Functional Tests, Arch. Otolaryng. **7**:606 (June) 1928.

conveying canal should be 3 mm. in diameter. The acoustic phenomena are those of a short tube closed at either end by a vibrating piston, and have been treated theoretically by Crandall.[27]

Now consider ankylosis of the stapes with reference to sound transmitted through the external canal. The fitting of the otosclerotic patient with a hearing aid is best done by means of either a bone oscillator or a magnetic air receiver connected with an individual ear mold having a long tip. The latter adjustment will be discussed. It is impossible to establish the diagnosis of ankylosis of the stapes clinically, and in recent literature it has been doubted on a statistical basis that ankylosis of the stapes is responsible for the impairment of hearing in otosclerosis.[28] Without entering into discussion one may assume that the fixation of the stapedial plate in the oval window is the chief cause of the impairment of hearing in otosclerosis. In the case of complete ankylosis of the stapes one is dealing with the problem of the reaction of a vibrating system on its support. The place of transmission to the support is the incudostapedial articulation and the support for the vibratory system of the middle ear in this case is the head of the stirrup bone. The vibrations from the receiver are transmitted over the closely coupled system in the external canal to the joint. One is dealing with bone conduction in this case in spite of the fact that an air conduction receiver is used. The experiments of Pohlman [31] prove that with reference to need of energy the promontory is of all places the most convenient for the application of a bone oscillator. He connected a thin bamboo rod with a bone oscillator and placed it on the promontory. In this place less output of energy was needed to get appropriate sound sensation than in any other place on the head.

The fact that an air conduction receiver may be used as a bone oscillator does not mean that bone oscillators of present design should not be used. There are conditions in the middle ear in which the bone oscillator is the specific receiver.

While most pathologic changes affect the vibratory system by increasing its stiffness, an interesting change should be mentioned which seems to be connected with lowering the stiffness. The associated symptoms of facial paresis permit such an assumption. In 1820 Roux, according to Landouzy,[32] observed in himself as a reaction to louder sound a "painful flutter of the drum." Landouzy [32] described hyperacusis in several cases. In his report he claimed that the exploring sound for the establishment of hyperacusis must be the louder the older the facial paresis. This is an interesting instance of a "loudness balance test" in the early days of otology. Moos [33] and Lucae [34] observed higher sensitivity toward low frequencies, which is in line with the lowered stiffness. In 1845 Wolff,[35] discussing the same disability, gave an explanation which sounds modern. He pointed out that the hyperacusis present in the beginning stage of the disability changes later into a "nervous hearing impairment" because the muscles of the middle ear lose their

27. Crandall, I. B.: Theory of Vibrating Systems and Sound, New York, D. Van Nostrand Company, 1926, pp. 98-102.

28. Weber, M. H. E.: Otosklerose und Umbau der Labyrinthkapsel, Leipzig, Poeschel & Trepte, 1935, p. 26.

29. Footnote deleted by author.

30. Footnote deleted by author.

31. Pohlman, A. G.: Further Objections to Accepted Interpretations of Cochlear Mechanics, Arch. Otolaryng. **35**:613 (April) 1942.

32. Landouzy: Compt. rend. Acad. d. sc. **31**:717, 1850.

33. Moos, S.: Ztschr. f. Ohrenh. **8**:221, 1879.

34. Lucae,[11] p. 356.

35. Wolff, H., in Lincke, C. G.: Handbuch der theoretischen und praktischen Ohrenheilkunde, Leipzig, J. C. Hinrichs, 1845, vol. 3, p. 85.

sound protective property. Contraction of these muscles increases the stiffness of the system. The result can be observed by persons who are able to contract the muscles of the middle ear voluntarily. If this is done in the presence of noise, the low frequencies disappear, the pitch of the noise is raised, and the loudness decreased. Experiments on animals [36] support the clinical observation.

The difference in perception of speech over the telephone between patients with middle ear impairment and those with perceptive loss is of clinical interest. It has been observed that patients with middle ear impairment hear well over the telephone but experience difficulty in understanding ordinary conversation at close range, whereas patients with perceptive loss are much more handicapped in hearing over the telephone than in hearing unaided conversation in a quiet room. The difference of reaction to conversation over the telephone has been observed with such regularity that it is believed to be of high diagnostic value. The question, "How do you hear over the telephone?" should be included in the history of every hard of hearing person. It gives a clue to the type of hearing impairment rather than to its degree. This opinion differs from Beasley's.[37] He classified the degree of hearing loss by judging the ability to hear over the telephone. To explain the aforementioned phenomenon, the properties of the telephone with reference to deteriorated hearing will be discussed shortly. The telephone distorts sound in a linear and in a nonlinear manner. Low frequencies are transmitted better than high ones, and the carbon microphone and the magnetic receiver are chiefly responsible for the nonlinearity. Now compare these properties with the requirements and properties of middle ear and of perceptive hearing impairment, respectively. (Middle ear hearing impairment: Increased power in transmission of the low partial frequencies is required. There is high tolerance to nonlinear distortion. The hearing aid with the highest degree of nonlinearity is a carbon microphone coupled with a magnetic receiver over a so-called "booster," a mechanical amplifier. It can be observed that some persons with middle ear disease understand speech better over this arrangement than persons with normal function. Perceptive hearing loss: Attenuated transmission of low partial frequencies and high fidelity in transmission of the high ones are required. There is low tolerance to nonlinear distortion.) Hence the different reaction toward speech over the telephone in different types of hearing impairment is readily understood. The amplification arrangement of present design provided by the telephone company is of use only for patients with prevalent middle ear hearing loss; it is of no use for persons with perceptive hearing loss.

Trying to explain the high tone loss in some types of middle ear impairment, Luescher [38] experimented by weighting the drum in different ways. The resulting hearing loss in the higher frequencies was given by him as a possible explanation for the high tone loss [39] in actual middle ear disease. The investigator cannot be agreed with here, because such experiments do not imitate sufficiently the actual pathologic conditions. An audiogram of a normal ear in which the external canal is filled with water is not convincing because neither the density of the water nor the boundary conditions between the air and the water have been taken into consideration. The audiogram taken on weighting the drum with mercury shows

36. Wever, E. G., and Bray, C. W.: (*a*) Ann. Otol., Rhin. & Laryng. **46**:947, 1937; (*b*) J. Exper. Psychol. **31**:35, 1942.

37. Beasley, W. C.: J. Motion Picture Engin. **35**:59, 1940.

38. Luescher, E.: Acta oto-laryng. **27**:250, 1939.

39. Crowe, S. J., and Guild, S. R.: Acta oto-laryng. **26**:138, 1938.

how additional mass changes the sound transmission characteristics of a vibrating system in an anticipated manner.

Not all symptoms of middle ear impairment of hearing can be explained by assuming that the ossicles and the drum are a simple vibrating system. Some cannot be explained at all with present knowledge of the physics of the ear. Some symptoms permit the assumption of a pathologic nonlinear distortion in the ear. The latter may be considered for a moment with reference to the fitting of hearing aids. Békésy [40] pointed out that pathologic changes in the middle ear may increase considerably the nonlinear response. Experimental interference with the middle ear shows similar results.[40a] Some symptoms can be observed which permit the assumption that they are caused by this change. It is well known that patients suffering from unilateral middle ear disease complain that an environmental noise well tolerated by the normal ear creates intolerable loudness in the affected ear. The best observers of the quality of sound, musicians, when suffering from the same disease complain of the roughness of the sound in the affected ear as compared with the sound in the unimpaired ear. Direct speech is heard as though it were transmitted over the telephone or the radio. Another symptom of nonlinearity, perceptive loss excluded, is the best understanding of speech at a particular level of speech power. Speech at a higher level of power is not understood, whereas it is well perceived by the unimpaired ear. Fletcher [41] explained a similar phenomenon in a normal ear as due to nonlinearity of the ear. Pure middle ear hearing impairment may sometimes be of the variable type, like the perceptive hearing loss, which is also of the variable type. Lorente de Nó [42] explained the phenomenon, discovered by Fowler, by recruitment, assuming that the variable type hearing loss is not encountered in middle ear impairment of hearing. Steinberg and Gardner,[43] on the other hand, state that

> . . . the philosophy underlying the method of loudness calculation suggests that nerve atrophy or nerve lesions would result in the variable type loss, and it would be expected that a middle ear or conductive lesion would result in the constant type loss. It cannot be concluded at present that such a state of affairs exists, because several cases having mixtures of constant and variable type losses have been found, which showed normal bone conduction thresholds.

Since bone conduction in these and in similar cases is not impaired in the higher frequencies, there is at the present stage of knowledge no reason to assume perceptive hearing loss, and therefore an explanation other than that of recruitment must be advanced. It is assumed that the pathologic loudness depends on the pattern of stimulation of the basilar membrane as well as on the total number of active neuroacoustic elements. The pattern of stimulation depends not only on the pressure spectrum in the external canal but also on the nonlinear distorting sound transducers in the ear. Hence pathologic loudness sensation can be explained as a function of the total number of available neuroacoustic elements with recruitment or as a function of the departure from the normal pattern of stimulation of the basilar membrane—assuming a normal total number of neuroacoustic elements—with pathologic nonlinearity in the ear.

Discrimination of symptoms on the basis of physical analysis has practical repercussions. If middle ear disease were obstructive in character only, it would

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40. von Békésy, G.: Akust. Ztschr. **4**:250, 1939.

40a. Neuman, E. B.; Stevens, S. S., and Davis, H.: J. Acoust. Soc. America **9**:107, 1937. Wever and Bray.[36b]

41. Fletcher, H.: J. Franklin Inst. **196**:289, 1923.

42. Lorente de Nó, R., in discussion on Fowler.[19]

43. Steinberg, J. G., and Gardner, M. B.: J. Acoust. Soc. America **9**:11, 1937.

be difficult to understand why some patients with middle ear disease can be fitted with a bone oscillator only, an air conduction receiver causing serious discomfort. This phenomenon can be explained, however, by taking pathologic nonlinearity of the sound transducers in the ear into consideration. In order to diagnose pathologic nonlinearity, loudness balance tests between bone and air conduction are proposed, besides the study of clinical symptoms. These tests cannot be performed, however, as long as the bone oscillators of audiometers are not calibrated in reliable units. The problem of calibration has been solved with elegance by Békésy.[44] The solution has not yet been applied to commercially available audiometers.

SUMMARY AND CONCLUSIONS

The individual ear mold of present design influences the transmission of sound from the receiver to the ear by attenuating higher partial frequencies. It represents a finite acoustic low pass filter.

The design of mold for middle ear impairment of hearing should be different from that for perceptive hearing loss. The mold for the former disability should have a long tip, and the diameter of the sound-conveying canal should be not less than 3 mm. The mold for perceptive hearing loss, on the other hand, should have a short tip, and the sound-conveying canal should be as large and straight as possible.

For the fitting of a patient who has perceptive hearing loss, an acoustic high pass filter is introduced in the form of a short tube with a side branch orifice. The advantages of this arrangement as compared with its electrical equivalent are outlined.

It has been shown for ankylosis of the stapes that sound transmitted from an air conduction receiver acts as if transmitted to the ear by bone conduction.

Middle ear impairment of hearing is not always of the obstructive type exclusively, but produces symptoms which strongly suggest pathologic nonlinear distortion of the sound transducers in the ear.

The study presented proves again that the fitting of hearing aids can be done only by persons who are trained in establishing the functional diagnosis of the ear and in basic acoustics.

The rational fitting of hearing aids not only is based on known symptoms of disease of the ear but enriches the symptomatology of disease of the ear.

NOTE.—Since this paper was accepted for publication, acoustical laboratory proof has been provided for the clinical experiments. The receiver with the mold was attached to an artificial ear of special construction. It could be shown on the tapes of a high speed level recorder that the dimensions of the sound-conveying canal in the mold as well as the described acoustic high frequency pass filter have a remarkable influence on the acoustical performance of hearing aids. The acoustical laboratory results will be published in a separate paper by F. M. Grossman and Charles T. Molloy.

APPENDIX

On the occasion of the review of the literature attention was drawn to J. G. Duverney (1648-1730). This great French physician seems to have been the first otologist who sought cooperation from a physicist, Mariotte, for the purpose of research in the field of the physiology and the pathology of hearing.[45] In his book, "Traité de l'organe de l'ouïe," which was published in 1683 (Paris, E. Michallet),

44. von Békésy, G.: Akust. Ztschr. **4**:113, 1939; J. Acoust. Soc. America **11**:259, 1939.

45. Politzer, A.: Geschichte der Ohrenheilkunde, Stuttgart, F. Enke, 1907, vol. 1, p. 201.

certain statements throw an interesting side light on the history of otologic research. The quotations which follow are from an early English translation.[46] (The sideheads are mine.)

Resonance of the External Canal.

. . . The obliquity of the passage is proper to preserve the membrana tympani from injuries of the weather and to render the vibrations stronger by doubling its refractions.

Sound Transmission Through the Middle Ear.

. . . There is room to believe that the air which is found in the tympanum being agitated by the pulsations of the membrana tympani, contributes partly at least to communicate them to the immediate organ. But nevertheless there is not apparent reason to imagine that the small quantity of agitated air is capable of shaking the os petrosum or rather the labyrinth which is contained in the os petrosum strong enough: So that we may assert with great probability that the pulsations on the membrana tympani are also communicated to the malleus, which the malleus communicates to the incus, the incus to the stapes whose vibration shakes the os petrosum and the labyrinthus . . .

Function of the Muscles of the Middle Ear.

. . . Is it the will that makes them act? There is not the least reason to suspect it. For in short, a noise most commonly surprises us without our being appriz'd of it. And it is my opinion that the objects themselves are the sole motives which cause these muscles to extend or relax the membrana tympani on different occasions, according to their impressions.

Resonance Theory of Duverney.

. . . A considerable branch of the portio mollis of the auditory nerve, after it is come to the basis of the cochlea, divides itself into many small ramifications, which passing through all the little perforations in the axis are distributed and lost in the different windings of the spiral lamina. In short, this lamina is not only capable of receiving the vibrations of the air but its structure makes it appear credible that it is sufficient to answer to all their different characters: For it being larger at the beginning of its first convolution than it is at the extremity of the last, where it finishes as in a point, and its other parts diminishing in proportion in bulk, we may suppose that the larger parts may be vibrated without the others participating in that vibration, and therefore they are capable of receiving none but the slower undulations which consequently answer to grave tones: and that on the contrary, its narrower parts being agitated, their undulations are quicker and consequently answer to acute tones in the same manner that the larger parts of a steel spring form the slowest undulations and answer the grave tones and so its narrower parts form quicker and more frequent vibrations, and consequently answer to acute tones. So that in short the spirits of the nerve, which expanded over its substance, receive different impressions which represent in the brain the different appearances of tones according to the different vibrations of the lamina spiralis.

Classification of Tinnitus.

We may after this manner lay down two sorts of noises in the ear, one of which proceeds from the disorders of the brain, the other from the disorders of the ear. Those which succeed the disorders of the ear are either true or false.

133 East Fifty-Eighth Street.

46. Duverney, J. G.: Treatise on the Organ of Hearing, Containing the Structure, the Use and the Disease of all Parts of the Ear, London, S. Baker, 1741.

INCIDENCE OF PATHOLOGIC SPEECH BEHAVIOR IN THE AMERICAN GENERAL POPULATION

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Otolaryngologic literature is completely lacking in recorded measurements of the incidence of pathologic speech behavior except for the statistics reported by Jackson and Jackson [1] on dysphonia plicae ventricularis within their practice on hoarseness, Voelker's [2] statistics on the same defect in the general run of people and Chervin's [3] surveys of speech defects among soldiers.

"Imperfection in articulate speech" [4] occurs in other forms than phonation with the ventricular bands. It is manifested in aphasia, paralysis, dysphonia, disorders of fluency, intonation and articulation and defects arising in amentia and psychoses.

This paper will attempt to report the incidence of imperfection in every phase of speech and voice in a carefully built-up sample numbering a hundred thousand people with a typical distribution of population characteristics in accordance with the scientific classification and nomenclature of the American Speech Correction Association,[5] in order to make such information available to writers in the field.

GENERAL CONSIDERATIONS

Procedure.—Six methods of examination were used: 1. Each subject was given a connected pronunciation (sound) test wherein his articulation (movements) was studied visually (labiomancy) for each phonetic element as he read a passage of an English test. 2. Each was given a standard whisper test for hearing and checked for lip reading. 3. Each was given unexpected and rapid fire questioning for psychoneurotic manifestations. 4. Each was given a physiologic examination of the speech organs, since speech tolerance for anatomic anomalies differs from other otolaryngologic standards. 5. Each was asked specifically regarding the history of his speech performance. 6. Each answer was studied for defects of voice and intonation.

Time.—The examinations were of a survey sort, so that the time given to each subject was brief. However, any statement of the average time per subject would be misleading, as the time varied greatly from subject to subject.

The rationale of the survey was important. Each normal-speaking subject was given as much time and as detailed a physiologic examination as each abnormal one. This survey found data on tolerance for anatomic anomalies not usually reported for normal speakers.

The survey of so large a sample of the population required more than a decade for its completion. In fact, this report represents the expenditure of more hours of work than any previous report of the type.

1. Jackson, C., and Jackson, C. L.: Diseases and Injuries of the Larynx, ed. 2, New York, The Macmillan Company, 1942, p. 70.
2. Voelker, C. H.: Frequency of Hoarseness Due to Phonation with Thyroarytenoid Lips, Arch. Otolaryng. **36**:71-78 (July) 1942.
3. Chervin, A.: Begaiements et autres maladies fonctionelles de la parole, Paris, Sociéte des editions scientifiques, 1902, pp. 149-277.
4. Jackson and Jackson,[1] p. 334.
5. Robbins, S. D., and Stinchfield, S.: A Dictionary of Terms Dealing with Disorders of Speech, Boston, American Society for the Study of Disorders of Speech, 1931, pp. 5-6.

Size of Population Sample Surveyed.—A total of 101,020 subjects were examined individually. The results reported for this hundred thousand sample represent work done by me and by trained assistants under my close supervision. A study of the data indicates that the sample is far larger than necessary in that successive 25,000 person divisions are comparable. Even the results from 1,000 person divisions will correlate if these have been selected by scientific sampling procedures.

I make no claim to having studied an unusually large sample, although I believe that 101,020 is large enough to be adequate. Westergaard [6] surveyed 34,000 school children. Also reported in what Panconcelli-Calzia now calls Vox was Lindberg's [7] survey of 297,000 school children. And a later issue from the Hamburger phonetische Laboratorium contained von Scarbo's [8] survey of 231,000 school children. Conradi [9] surveyed 87,444 school children. Wallin [10] surveyed 89,057 school children. West,[11] with the help of Camp, Travis, Stinchfield and Twitmyer, surveyed 3,820,405 school children. Louttit and Halls [12] surveyed 199,839 school children. Carhart [13] surveyed 144,570 high school pupils.

Earlier Surveys.—This survey is the first and only one based on complete clinical technic and the scientific nomenclature of the American Speech Correction Association. It stands as the first and only survey of America's general population. It is the only survey reporting all ages. Previous studies, such as those mentioned in the foregoing section, were made by educators and by graduate students of speech and were entirely concerned with public school children. Several more studies of the same nature have been reported but deal with far fewer subjects.[14] It will be noted that these are surveys of public school children and do not attempt to represent the case material of otolaryngologic practice.

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6. Westergaard, H.: Von der Häufigkeit der Sprachgebrechen, Med.-pädagog. Monatschr. **8**:1-8, 1898.

7. Lindberg, K.: Zur Häufigkeit des Stotterns bei Schulkindern, Med.-pädagog. Monatschr. **10**:281-286, 1900.

8. von Scarbo, A.: Statistik der an Sprachstörungen leidenden Schulkinder Ungarns, Med.-pädagog. Monatschr. **11**:65-89, 1901.

9. Conradi, E.: Speech Defects and Intellectual Progress, J. Educ. Psychol. **3**:35-38, 1912.

10. Wallin, J. E. W.: Speech Defective Children in a Large School System, Miami Univ. Bull. **25**:45 (Dec.) 1926.

11. West, R.; Camp, P. B.; Travis, L. E.; Stinchfield, S. M., and Twitmyer, E. B.: The Defective in Speech, White House Conference on Child Health and Protection, III F, Special Education: The Handicapped and the Gifted, Committee on Special Classes, New York, 1931, pp. 349-384.

12. Louttit, C. M., and Halls, E. C.: Survey of Speech Defects Among Public School Children of Indiana, J. Speech Disorders **1**:73-80, 1936.

13. Carhart, R.: A Survey of Speech Defects in Illinois High Schools, J. Speech Disorders **4**:61-70, 1939.

14. West, R., and others: J. Speech Disorders **4**:356, 1939. Root, A. R.: A Survey of Speech Defectives in the Public Elementary Schools of South Dakota, Element. School J. **26**:431-441, 1926. Blanton, S.: A Survey of Speech Defects, J. Educ. Psychol. **7**:581-592, 1916. Young, J. A.: Speech Rehabilitation in the Rural Schools of Waukesha County, Wisconsin, J. Speech Disorders **5**:25-28, 1940. Tucker, A. C.: Impediments of Speech in Children, Education **34**:354-360, 1914, Rouma, G.: Enquete scolaire sur les troubles de la parole chez les ecoliers belges, Internat. Arch. f. Schulhyg. **2**:151-190, 1906. Smith, L. L.: A Survey of Speech Defective and Behavior Problem Students in Two Elementary Schools, Thesis, Northwestern University, 1938. Snow, L. C.: A Survey of Speech Defects in the Granite School District, Utah, Thesis, University of Utah, 1935. Dollard, M. M.: A Survey of the Speech Disorders of the Children in the Catholic Parochial Schools of Madison, Wisconsin, Thesis, University of Wisconsin, 1925. Francis, J. T.: A Survey of Speech Defects of Iowa City, Iowa, Thesis,

(Footnote continued on next page)

Severity of Defect Reported.—This report is not primarily concerned with minor speech defects which are no more than quasihandicaps of personality and which would not cause patients to seek out the help of otolaryngologists. Such defects make up the vast majority of the speech handicaps which are the subject of educational literature and of psychiatric behavior problem clinics. The records of this survey indicate that these minor defects handicap approximately 120 per thousand of this population sample. Persons thus defective are referred to as the ones hard to understand or the ones with unpleasant voice.

Instead, this report is primarily concerned with those who are suffering from pathologic conditions of so severe a degree as to disable them and render them more or less helpless in society as far as speech and voice are concerned and who tend to develop vicarious means of communication or to enlist the aid of their fellow men. When rounded to two significant figures, this survey indicates that the otolaryngologist can expect to find 30 such people per thousand of population. This is shown in table 3. For comparison, the minor defects are included in tables 4 and 5.

Type of Subject Examined.—According to W. H. Sewell, the sociologist, the term "general population" is used to designate the total number of persons living in the United States without respect to the composition of the group. Sewell also holds that in order to demonstrate the representativeness of the sample studied, it is sound procedure to compare the characteristics of the selected population with those of the general population as given by the census report. It is held that since the data on this hundred thousand sample are comparable with the data of the 1940 United States Census to a degree slightly over 90 per cent they can be used for prediction of the incidence of each type of speech defect in the American general population.

Sex.—This survey included the examination of 73,745 males and 27,275 females. The nation as a whole in 1940 was made up of 66,061,592 males and 65,607,683 females.

Age.—The average age of this sample was seven years less than that of the general population. Whereas the present sample had an average age of 20, the nation's average age was 27 years. In this sample 55.1 per cent were under 25

State University of Iowa, 1930. Hiser, N. O.: Preliminary Survey of Speech Defects Among Freshmen at the University of Illinois, Thesis, University of Illinois, 1926. Varnado, N. M.: A Survey of Speech Defects in the Grammar Schools of Baton Rouge, Louisiana, Thesis, Louisiana State University, 1937. Tanberg, M. C.: A Study of the Incidence of Speech Defects Among Children in the State of Washington, Thesis, University of Washington, 1937. Caldbick, H. M.: A Survey of Speech Defects of the Grade School Children in the Public Schools of Everett, Washington, Thesis, University of Washington, 1937. Blyton, G. S.: A Survey of Speech Defects in the Public Schools of Puyallup, Washington, University of Washington, 1937. Eiseman, C. L.: Survey of Speech Disorders Found in the Catholic Parochial Schools of Madison, Wisconsin, Thesis, University of Wisconsin, 1925. Mills, A. W., and Streit, H.: Report of a Speech Survey, Holyoke, Massachusetts, J. Speech Disorders **7**:161-168, 1942. Burt, C.: The Backward Child, New York, D. Appleton-Century Company, Inc., 1937, pp. 165-166. Evans, D. R.: Report of Speech Survey in the 9-A Grade, Quart. J. Speech **24**:83-90, 1938. Morris, D. W.: A Survey of Speech Defects in Central High School, Kansas City, Missouri, ibid. **25**:262-268, 1939. Rogers, J. F.: The Speech Defective School Child: What Our Schools Are Doing with Him, Bull. 7, United States Department of Interior, Office of Education, 1931, p. 5. Steer, M. D., and Trimble, D. C.: Speech Handicaps of Students in Purdue University, Bull. Purdue Univ. Stud. in Higher Educ. **28**:17 (April) 1936. McAllister, A. H.: Clinical Studies in Speech Therapy, London, University of London Press, 1937, pp. 7-15. Burden, L. G.: A Survey of Speech Defectives in the Indianapolis Primary Grades, J. Speech Disorders **5**:247-258, 1940. Carrell, J. A.: A Comparative Study of Speech Defective Children, Arch. Speech **1**:179-203, 1936.

years of age, whereas in the nation the percentage was 43.2. Table 1 shows a detailed age comparison of the present sample with the whole population as reflected in the 1940 census.

Race.—This sample contained a higher percentage of white persons (99.6) than was found in the nation in 1940 (89.8 per cent of the white race and 10.2 per cent of other races).

Rural-Urban Ratio.—Whereas the nation in 1940 was divided into 74,423,702 urban and 57,245,753 rural persons, the present sample was divided into 74,399 urban and 26,621 rural persons.

TABLE 1.—*A Comparison of the Sample Examined (101,020 Persons) with the Nation as a Whole as Described in the 1940 United States Census by Age Group (Given in Percentages)*

Age Group	This Study	1940 Census
Under 5 years	0.5	8.0
5 to 19 years	49.4	26.4
20 to 24 years	5.2	8.8
25 to 49 years	21.8	36.4
50 and above	13.1	20.4

TABLE 2.—*A Comparison of the Distribution as to Type of Work of the Employed Subjects Examined in This Survey with That of the Employed in the Nation as Given in the 1940 United States Census (in Percentages)*

Type of Work	This Study	1940 Census
Agriculture and like industries	44.1	18.8
Mining	2.0	2.0
Construction	3.0	4.6
Manufacturing	27.7	23.4
Transportation	2.3	6.9
Repair	0.1	1.9
Personal service	0.1	8.9
Finance	5.9	3.2
Recreation	0.1	0.9
Professional service	10.4	7.3
Government	1.7	3.9
Wholesale, retail	20.8	16.7
Total	100.0	98.5 *

* One and one-half per cent were not comparable. For complete description of what these employment classifications include, see detailed census reports.

Employment.—In order to show the employment status of the people examined in this study, it was necessary to eliminate all unemployed people. So, 304 subjects who were orphans, institutionalized patients or invalids, 1.2 per cent in nursery schools and kindergartens, 35.0 per cent in elementary schools, 8.2 per cent in secondary schools and 10.5 per cent attending institutions of higher education were substracted. Discounting for housewives and unemployed persons above 14 years of age, then, leaves 39.3 per cent of those examined in the gainfully employed class. Table 2 shows the distribution by type of employment and how this compares with that from the 1940 census.

Geographic Distribution.—The subjects were surveyed in the states of Ohio, New Hampshire, Vermont, Iowa and Oklahoma. They came from home towns

and had lived in places located in all forty-eight states and Alaska, although not in appropriate numbers. The subjects in this sample spoke dialects of English including all major American regional variants: general American, southern American and eastern American.

Types of Pathologic Speech Surveyed.—It was not possible to survey all aspects of language behavior, but all types of defective speech as listed by the American Speech Correction Association were included, as follows:

1. Dysphasia. Persons with this defect are characterized by an inability to comprehend or to formulate speech, and there are expressive, receptive and amnesic types. All show partial destruction of the dominant frontal lobe of the cerebrum together with lesions of the posterior lobe of varying extents.

2. Dysarthria. This includes all disturbances of articulation due to lesions interfering with the discharge of the motor impulse through the pyramidal, extrapyramidal and cerebellar tracts or through the final common neurons the nuclei of which are in the bulb. The paralysis of speech has the same characteristics as does the general paralysis, and flaccid, ataxic, athetoid (including rigid and torsive) and spastic forms occur.

3. Dysphemia. This term is given to all speech disturbances regardless of form which arise as stigmas of psychoneuroses. It is illustrated as spasmophemia, cryptogenetic spasmophemia, aphemia and tachyphemia.

4. Dyslogia. This is a weakened form of language behavior due to an impairment of reasoning resulting from amentia or semantic disorders.

5. Dyslalia. This is a defect of articulation dependent on malfunctioning of the lips, the tongue or the palate or on a lack of compensation for malformation of these articulators. It is manifested in brogues, phonetic defects, hearing defects, short span of auditory memory, lalling, delayed speech, baby talk, invented language and lisping.

6. Dysrhythmia. This defect of intonation is manifested as defective breath grouping, defective placement of stress, pitch pattern perseverations and defective syllabic rate.

7. Dysphonia. This term includes defects of voice caused by neuronal lesions resulting in paralysis of the vocal cords, hysterical conditions manifested in loss of voice, phonasthenia and bizarre qualities, and organic alterations resulting from excision of the vocal cords, laryngectomy and presence of an interlaryngeal web. It also includes defects of voice associated with short palate, long palate and cleft palate. There are functional nasality and denasal voice. Occupational disease and irritants result in hoarseness. There is hoarseness due to constriction from the superior thyroarytenoid lips and the sphincter formed by the aryepiglottidean folds, together with the pulvinar of the epiglottis and the eminences of Wrisberg and Santorini. These abnormalities are demonstrated in infantile voice, strident voice, ventricular voice, flat laryngeal twang, gravel voice and breathy voice. There are also pharyngeal guttural voice and buccal mushy voice.

INCIDENCE OF PATHOLOGIC SPEECH BEHAVIOR

It was found that 30 per thousand of the sample had defects. Some subjects had more than one defect. Thus, in the general population 30 per thousand can be expected to have disabling disturbances of speech. There will be 1.1 with dysphasia, 2.9 with dysarthria, 9.1 with dysphemia, 5.0 with dyslogia, 7.2 with dyslalia, 4.4 with dysrhythmia and 9.7 with dysphonia per thousand people.

It was found that there are to be expected 120 minor speech handicaps per thousand. There are to be expected 2.1 with dysphasia, 4.2 with dysarthria, 27.1 with dysphemia, 37.2 with dyslogia, 93.7 with dyslalia, 18.5 with dysrhythmia and 49.9 with dysphonia per thousand people in the general population.

TABLE 3.—*Incidence of Serious Disturbances of Speech* *

Type of Pathologic Condition	Frequency of Occurrence, per Cent	Relative Frequency, per Cent
Dysphasia (weakened mental imagery)	0.11	2.8
Dysarthria (paralyzed speech)	0.29	7.3
Dysphemia (intermittent nervous disorders)	0.91	23.1
Dyslogia (difficulty in expression of ideas)	0.50	12.7
Dyslalia (defects in articulation)	0.72	18.3
Dysrhythmia (defects of rhythm)	0.44	11.1
Dysphonia (disturbances of voice)	0.97	24.6
All types	3.00 †	99.9

* This table gives the incidence of disabling abnormalities of speech and voice in America's general population as predicted from a sample (101,020 subjects) with population characteristics (age, sex, race, occupation, school, rural and urban distribution) controlled so as to be comparable within 90 per cent with the 1940 United States census.

† Some subjects had more than one defect. This figure is rounded to statistically significant integers.

TABLE 4.—*Incidence of Minor Speech Defects* *

Type of Defect	Incidence, per Cent	Relative Incidence, per Cent
Dysphasia (weakened mental imagery)	0.21	0.9
Dysarthria (paralyzed speech)	0.42	1.8
Dysphemia (intermittent nervous disorders)	2.71	12.0
Dyslogia (difficulty in expression of ideas)	3.72	15.9
Dyslalia (defect in articulation)	9.37	40.2
Dysrhythmia (defect of rhythm)	1.87	8.0
Dysphonia (defect of voice)	4.99	21.4
Prevalence of defects	23.29	
Number of defective subjects	12.00 †	100.0

* This table contains an estimate of the prevalence of minor handicaps of speech and voice which form problems of school physicians and nurses, psychiatric and psychologic behavior guidance centers, kindergarten and elementary school teachers, high school and college speech teachers and industrial rehabilitation departments, which are manifested as minor complications and sequelae of disease, behavior problems, lack of compensation for anatomic anomalies and lack of training and which serve as nuisances and as limitations on personality. This estimate for the American general population is predicted from a sample numbering a hundred thousand subjects so selected as to age, sex, race, occupation, school grade and rural or urban residence as to be comparable with the nation as a whole.

† Some subjects had more than one defect. This figure is rounded to statistically significant integers.

TABLE 5.—*Total Incidence of Subnormal Language Behavior; Distribution of Both Handicapping and Disabling Speech and Voice Disorders in the American General Population*

Type of Disorder	Predicted Frequency, per Cent in General Population	Relative Frequency, per Cent
Dysphasia (weakened mental imagery)	0.32	1.2
Dysarthria (paralyzed speech)	0.71	2.6
Dysphemia (intermittent nervous disorders)	3.62	13.3
Dyslogia (difficulty in expression of ideas)	4.22	15.5
Dyslalia (defect in articulation)	10.09	37.1
Dysrhythmia (defect of rhythm)	2.29	8.4
Dysphonia (disorders of voice)	5.96	21.9
All types	15.00 *	100.0

* Some subjects had more than one defect. This figure is rounded to statistically significant integers.

IMPLICATIONS AND INDICATIONS

The rationale of examining the normal speakers with the same elaborate care that was given to the subnormal ones led to additional findings of importance, as follows:

1. One person having a cleft palate was found without a speech defect, and others, with only minor problems.

2. Laryngectomized persons were examined whose speech was peculiar, but 2 others could in no way be called defective.

3. Tongue-tie was found to have no relation to speech performance. All 5 of the persons with severe tongue-tie demonstrated no speech difficulty. Over a hundred whose ties had been clipped had speech defects. Many had minor ties with no defects. It seems that Spencer and Cade [15] are correct in their recommendation that the clippers be punished. It is unfortunate that even the pediatric texts of the last three years perpetuate this four hundred year old medical superstition.

4. The most perfect dentition occurred with a severe lisp. This photographically unretouched girl had too even a bite to speak a language invented by a race of notched teeth people. But others with teeth almost as perfect had no "s" trouble.

5. One subject was found who complained of having difficulty in making people understand him. It was found that the opening between his lips did not correspond with the opening between his teeth. When he opened his lips a half inch, he displayed the upper part of the maxillary teeth and the lower part of their gums. He could not be said to have an abnormally short upper lip, but he did have a projecting mandible.

6. Four deaf people with normal speech were found. This is not many if one considers how far the science of aiding deaf persons has advanced. On the other hand, this survey considered such minor things as characteristic deaf voices, the correction of which is too new to show results. The results of this survey indicate that a voice test would be a reliable hearing test. It seems that the voice sign is the first diagnostic sign of hearing loss. This should prove of great value in preventing deafness in that it will make possible the early catching of the curable types. It was found that the speech defects of the hard of hearing were correctable. Certainly lip reading is a success. And certainly some few ear phones are already completely satisfactory.

7. Twenty-four hundred persons who formerly stuttered were found, illustrating the absolute curability of the defect. If the defect was overcome in childhood, the memory of it was completely gone. If it persisted until it modified social status and life goals, the kinesthetic memory may have been completely forgotten, but the psychologic limitations and social tortures may be vividly retained. Outgrowing methods, self-cure methods, haphazard methods, superstitious methods, educational methods and professional methods seemed to have worked with complete success, and, unfortunately, it could not be shown that clinical procedures were the only efficacious ones.

8. Twenty-seven subjects who had had dysphasia seemed to be quite normal. Of course, it is difficult to know how restricted they had been during their speech disorder. And it is impossible to compare their behavior before and after in this survey, which is not surprising to any one familiar with Brickner's [16] research on the subject.

9. An inspiring goal was set by the recovery of speech in paralysis. Eight subjects were found who, even in the facial display of emotions, did not give away their history. One admitted self consciousness about speech when overfatigued.

15. Spencer, W. G., and Cade, S.: Diseases of the Tongue, Philadelphia, P. Blakiston's Son & Co., 1931, pp. 17-20.

16. Brickner, R. M.: The Intellectual Functions of the Frontal Lobes, New York, The Macmillan Company, 1936, p. 354.

10. In general, it seems that in the cases of dysphasia and dysarthria the practice has been to leave the patient resistant to general disease, but helpless or handicapped, and left to his own resources for vocational rehabilitation.

11. The subjects with cryptogenetic spasmophemia presented an interesting picture. These psychoneurotic persons talked normally except for a 45 year old foreman, who had dysgrammaphemia. All were full of self pity for themselves because they could not bring themselves to utter, and had to substitute for, their peculiar taboo words.

12. This survey had many results promoting health and was socially valuable beyond the recording of pure research data. One subject was reported by the Boston General Hospital to have been caught early enough to bring relief from schizophrenia. The number of persons with hearing problems caught at an early stage, who were referred to otologists and given relief, will many times repay society for the small investment in this survey.

13. Little positive result was demonstrated by the survey drawing the attention of educators to the problem except in passive interest.

14. The tolerance of the subject for articulatory disorder was beyond all expectation. The most bizarre defect seemed to be suffered without strain or nervousness or consciousness. An articulatory disorder has to be very severe before it disables the subject.

15. Dysrhythmia leaves the subjects unsuspicious of the nature of their handicap except for defects of syllabic rate, about which they say, regardless of their slowness, that they talk too fast.

16. In general, popular articles on the subject of speech defects seem to have confused rather than elucidated the thinking of the sufferers. These articles have positive results in respect to combating the idea of incurability, but it is the general practitioner who has changed the construct that the baby was marked for defective speech.

17. To the population here surveyed, it is the thing to do to have childhood diseases; in their minds chronic respiratory disease is not worth medical attention. Certainly more education is needed for laryngology, such as is being undertaken by otologists.

18. Arrests of the development of speech for long periods up to 5 years of age as the result of disease, lack of training, social tensions and other circumstances have life-long sequelae.

19. An anatomic anomaly is not in itself a sufficient and adequate cause (in the mathematician's sense) to produce a speech defect. Too many normal speakers were found who had severe structural limitations to permit a division into organic and functional in the traditional sense. However, it appeared that a certain structural configuration in a subject in whom that structural configuration played an active part in speech did react in a way unique for that structural configuration and that this reaction correlated with traditional descriptions in the literature. It is believed that the speech therapeutist can learn many clinical devices by studying, with laboratory apparatus, how normal speakers have compensated for their deformities.

20. The aspiration of many general practitioners to be elite and practice surgery, especially the minor surgery of tonsillectomies, seems to have resulted in many structural defects. The number of persons who were examined with paralysis of the palate, enucleation of the uvula, scarred pharynx and other defects as the results of accidents during tonsillectomies surpasses all belief. The number

found in this study is of the order of thousands and is being reported separately. If the operation was completed with success, however, no great improvement in a speech defect seemed to have resulted, and no voice seemed to have been ruined.

21. The assertion that macroglossia and microglossia are causes of speech defects must have been the result of superstition or armchair philosophy rather than clinical observation.

22. Orthodontal appliances seem to improve rather than to obscure speech. After the removal of the wires, superior speech can be predicted.

23. Speech therapeutists should educate the public concerning intonation. Parents do not teach their children intonation. In fact, most often they suppress it in order to build vocabulary. The teaching of intonation in the schools is only misguidance.

24. In general, voice training seems to have been neglected, but the time for it is evidently at hand. "Too" terms should not be used by teachers or parents. Don't say, "Speak louder." And don't say, "Speak lower."

25. Attention to semantic disorders is much needed.

26. Articulation gymnastics should be administered on a national scale.

27. The poker face ideal and stance is a great handicap to the American's expression.

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HYGIENE OF THE VOICE

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Analysis of the voice of sucklings shows that they have two different cries; one has a soft and the other, a hard beginning. These cries were described in detail by Flatau and Gutzmann,[1] who expressed belief that the soft onset (*Einsatz*) is significant of a good mood while the hard one indicates distress. But frequently the cry begins softly and changes more or less rapidly into a hard one. This indicates that the words "soft" and "hard" describe not only the beginning, or onset, but some auditive characteristics during the continuation. The onset depends on the form of the glottis at the beginning of the cry.[2] Three possibilities exist: 1. The glottis may form a triangle, as it does in ordinary exhalation; in this case the cry or the vowel will be introduced by an *h*, which is nothing but an audible exhalation. 2. The vocal cords may approach each other to form a very narrow slit; in this case the beginning will be soft. There is only a gradual change from the first to the second form; by phonetic experiments it can be proved that the soft onset is also a simple exhalation preceding the production of voice, but this feeble exhalation is perceived by the ear not as a consonant but only as a soft onset. 3. If the glottis is closed firmly, the voice production is preceded by an explosion. This is called the hard onset, or the *coup de glotte*. But as the continuation of the voice (as well as that of a vowel) may also give the impression of softness or hardness, the quality evidently depends on circumstances other than the initial form of the glottis. One might assume that the return of the vocal cords to the same position as that at the onset could explain the continued impression of hardness or softness, but stroboscopic examination shows that during continual voice production the intimate closure, characteristic of the *coup de glotte*, does not occur, at least not rhythmically. The hard voice, therefore, depends on other functional characteristics, namely, hyperfunction of certain muscles in some of the upper resonating cavities.

I could distinguish six types of hyperfunction, with the locations shown in the scheme (figure). Type *1* is hyperfunction of the muscles for closing of the glottis, and is therefore identical with the *coup de glotte*. Consequently, this type does not involve the resonating cavities, but it may be mentioned here because, as will be seen later, there exists a functional unity between this first type of hyperfunction and the other forms. Type *2* might be called by a popular word, self strangulation. It consists of hypercontraction of the lower part of the pharyngeal cavity, due to hyperfunction of some pharyngeal constrictors. In type *3*, called the *Knödel* (dumpling) type, the listener gets the impression of a dumpling-like mass obstructing the throat. Type *4* consists of hypercontraction of the soft palate, producing in the listener the impression of lack of nasal resonance. (A permanent state of palatal hyperfunction is known as functional hyporhinolalia, or functional rhinolalia

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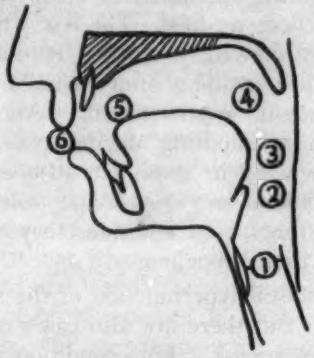
1. Flatau, T. S., and Gutzmann, H.: Die Stimme des Säuglings, Arch. f. Laryng. **18**: 139-151, 1906.

2. The expressions "soft" and "hard" are used also for the initial sound of a vowel in articulated speech.

clausa; the speech resembles adenoid speech, in which nasal resonance is prevented by the presence of these growths, which seem to act like a great number of cushions in a musical hall, damping the normal resonance of the room. In the condition under discussion this hyporhinolalia is not permanent, but appears temporarily during speaking or singing.) Type *5* consists of stiffening of the anterior part of the tongue, and type *6*, of stiffening of the lips.

A more precise description and a report of clinical experiences concerned with the various types of hyperfunction have been published.[3] I have already spoken of the functional unity which exists between the muscles closing the glottis and the upper resonating cavities. Experience shows that persons with *coup de glotte* present others of the five types of hyperfunction, and, further, that these five types exhibit a close relationship, which makes some of them appear simultaneously or successively in one and the same person. It is, however, possible for a trained person to produce a single one of the six types of hyperfunction without another.

The cry of the suckling which has a hard onset and continues to be hard is an example of the *coup de glotte* combined with hyperfunction of the upper resonating cavities. The most frequent combination of hyperfunctions is that of the first and



Locations for the six types of hyperfunction: *1*, the *coup de glotte; 2*, strangulated voice; *3*, the *Knödel*, or dumpling; *4*, palatal hyperfunction, or functional hyporhinolalia; *5*, stiffening of the anterior part of the tongue, and *6*, stiffening of the lips.

second types. My experience is that sucklings frequently begin with a soft cry, but slowly or rapidly change it into a hard one. This means that initial gentle closure of the glottis is followed by hyperfunction of some pharyngeal constrictor muscles.

If the cry is started as hard and is continued as hard, or if it is started as soft and is continued as hard, the cry is a sign of distress, or at least of an unpleasant emotional state. Unfortunately, nature cannot escape showing that sign of distress or unpleasant emotion in later life. A person in distress, in danger or in violent pain will, if he cries at all, use, involuntarily even, the hard form. More than that, every state of higher emotion is commonly associated with some form of hyperfunction of the voice and resonating apparatus. That is why a person with a voice which is not well trained "strangles" himself, and uses the hard voice at the onset, in public speaking, preaching, acting and commanding. But, in everyday speech, also, the sensitive ear will detect slighter or greater degrees of self strangulation. This may be due, at least in part, to a habit acquired by the frequent use of the *coup de glotte* and other forms of hyperfunction in public speaking, preaching and the like.

3. Froeschels, E.: Laws in the Appearance and Development of Voice Hyperfunctions, J. Speech Disorders **5**:1-4 (March) 1940.

If the voice in the speech of everyday life is restricted to shorter periods of strain only, no dangerous, or even unpleasant, consequences may arise. But if a person has to speak very much, as in the case of a salesman, a teacher or a lawyer, after a certain time symptoms and signs of voice trouble may appear. Singing is less harmless than speaking, even if used only for pleasure and relatively seldom. Such nonprofessional singers may acquire real voice trouble after some months or years. It is a matter of experience that an unhygienically used singing voice influences unfavorably also the speaking voice. The reverse is less frequent, but nevertheless possible, namely, that an overstrained speaking voice damages the qualities of the singing voice. I have described this disease as rhesiogenic phonasthenia.

The signs of an overstrained voice are much more frequent than the symptoms. Many school children, even preschool children, speak and sing with hyperfunction. At the beginning of my practice, I found in Vienna about 50 per cent with "hoarse" voices, but in the last years of my presence there I encountered 93 per cent. This, evidently, was accounted for by the greater acuity of my ear. Among the school children I made an observation which, again, proves the influence of emotion on the voice. I examined the voices of 1,000 school children while they were speaking and while they were reading. The few who did not show straining of the voice during speaking showed it during reading. The slight emotion produced by the difficulties which reading offers a child seemed to be one of the reasons for the sudden increase in hyperfunction.[4] Another source of the functional hoarseness of children is shouting during play, and, in particular, an unhygienic singing frequently used in grammar schools, at least in Europe, is detrimental to the voice. Without any systematic voice training children have to sing songs with too high tones. In addition, they try to sing very loud in order to be better heard than their schoolmates.

Thus far I have spoken only of hyperfunction of the muscles of the glottis and the upper resonating cavities. But there are also cases of functional voice trouble in which signs of hypofunction appear. This condition may deserve the name of myasthenia laryngis (Jackson[5]). The signs are not so clear as those of hyperfunction. I formerly called such trouble "paretic hoarseness," to differentiate it from hyperfunction, which I called "spastic hoarseness." Now I prefer to eliminate the word "hoarseness" entirely, for it does not contain any possibility of a finer differentiation. Such a differentiation is strongly indicated, for the attentive ear of the observer receives many different impressions from the voices of "hoarse" patients. The paretic, or hypofunctional, state may be characterized by relaxation of the muscles closing the glottis, and probably of some pharyngeal muscles also. It is hard to describe the auditive impression, but it is suggested that this hypofunctional difficulty resembles a voice troubled by the presence of much mucus in the larynx.

Development of functional voice troubles of this kind always begins with hyperfunction, which later may be complicated by hypofunction and finally may or may not pass into the hypofunctional state only. (If a functional voice trouble has been proved to have started not with hyperfunctional but with hypofunctional signs, it is psychogenic, and not the consequence of misuse of the voice.[6])

4. Hygiene of the voice, therefore, requires avoidance of hyperfunction in reading.

5. Jackson, C.: Myasthenia Laryngis, Arch. Otolaryng. **32**:434-463 (Sept.) 1940.

6. Froeschels, E.: Psychogenic Impediments of the Voice, Laryngoscope **49**:1225-1230 (Dec.) 1939.

It is not within the scope of this paper to discuss the exact symptoms of those functional troubles of the voice which are known all over the world as phonasthenia.[7] Only a few words may be devoted to this matter. In addition to the hyperfunctional and hypofunctional troubles, paresthesias, pain and a feeling of dryness may appear, which contribute to the always increasing nervousness of the patient. The diminution in the beauty of the voice and its efficiency is another factor which makes the patient suffer. As nervousness is frequently associated with hypertension, a vicious circle may be formed, consisting of the original hyperfunction and the hyperfunction resulting from the nervous tension.

It is, likewise, not within the scope of this paper to deal with the therapy of the initial hyperfunctional states and their sequelae. If I, nevertheless, undertake to speak briefly of some therapeutic means, it is because the hygienic measures evidently cannot be established, as a rule at least, before some abnormal habits of the voice apparatus have already developed. If the suckling already frequently uses the hyperfunctional cry, the time for real preventive hygiene of the voice is past. One can only prevent persistence and development of the signs of hyperfunction and their consequences. That is why, in my cases, hygienic measures and therapy are somehow identical, and I, therefore, may be permitted to say a few words about the treatment of functional voice troubles.

It is evident that the treatment of signs of hyperfunction and their accompanying symptoms must aim at elimination of the hyperfunction. On the other hand, the paretic signs demand a method of strengthening. The methods of treatment directed against hyperfunction are generally called, and are indeed, methods of relaxation. The whole literature of singing methods and of methods of training the speaking voice is full of advice on relaxation. To name a few of the strengthening procedures, I might mention the method of Arnim (*Staumethode*); the different methods of training in breathing which try, at least partly, to strengthen, with the respiratory muscles, other muscles of the singing and speaking apparatus; the method of Flatau [8] and that of Mithoefer,[9] in this country, which use the electric current on the larynx, and the employment of initial plosives to influence the laryngeal muscles by the strong impetus which the muscles of articulation exert if they themselves are in a state of strong contraction, as is characteristic for the production of plosives.

I have used many of these methods for many years. They have some advantages and some disadvantages. One of the chief disadvantages, in my experience, is the patient's difficulty in coordinating the different functions which he is taught—for instance, deep inhalation, thrifty exhalation and a relatively relaxed condition of the mouth. Therefore I have been searching for years for a simple method which combines all the functions involved in voice and speech production.

By many roundabout ways, I finally evolved the chewing method, a description of which was first published in this country.[10] The method is based on the obser-

7. Asthenia means weakness; the troubles of unhygienically functioning voices begin usually in the period of hyperfunction. The word "phonasthenia," therefore, should not be used for all the phases of trouble of unhygienically functioning voices. One should, rather, speak of hyperfunctional and hypofunctional voices with or without nervous sensations.

8. Flatau, T. S.: Die funktionelle Stimmschwäche (Phonasthenie) der Sänger, Charlottenburg, Germany, G. Bürkner, 1906.

9. Mithoefer, W.: A Simple Treatment for Defects of the Singing and of the Speaking Voice, Arch. Otolaryng. **31**:16-22 (Jan.) 1940.

10. Fröschels, E., and Jellinek, A.: Practice of Voice and Speech Therapy, Boston, Expression Company, 1941.

vation of everyday life that one can chew and speak (and even sing) at the same time. Since the same muscles and the same nerves of the upper resonating cavities are engaged in the two functions, I concluded that chewing and speaking must be somewhat identical. One cannot perform two different actions at the same time with one set of muscles. Further consideration and observation led me to conclude that chewing was the origin of human speech. Members of primitive tribes continually produce voice during chewing. I learned from the African traveler Czermak that if one approaches a village of the Fellahin, in the north of Egypt, one does not know whether they are eating or entertaining. My surmise is that with primitive man, hundreds of thousands of years ago, it was probably the same, and that he acted with even greater energy in chewing and voice production. Therefore there existed a primary function of origin for chewing and voice production. That may have been the reason that chewing movements accompanied voice production when, in any emotional state, primitive man shouted. The assumption that such was the case explains articulated speech. (Other authors face tremendous difficulties in their attempts to explain the movements of articulation. The bibliography on this subject may be found in the book just mentioned.[10])

Physiologic and pathologic observations seem to prove this theory of the origin of human speech. Mammals frequently produce voice while chewing. The babbling period of sucklings has been considered training for articulated speech, but it seems also to be training for chewing. Frequently, bad eaters are also slow in speaking, and some good eaters become bad ones when they begin to speak. Some aphasic patients, if they want to speak and cannot find words, do nothing but chew with voice production, and so do patients in the state of awakening from an epileptic seizure.

The most impressive experience to me has been the immediate change in congenitally deaf people from an abnormal to an almost normal voice. In the Central Institute for the Deaf, St. Louis, in the speech clinic of Ann Arbor, Mich., and in various countries in Europe, I have been able to demonstrate that fact in numerous pupils and in former pupils of schools for the deaf. If a person who has never heard speech suddenly acquires a pleasant voice when ordered to chew with voice production, it seems to indicate that chewing is the source of human speech, and that the association is still present and working if stimulated.

All these observations convinced me that the chewing method was the therapeutic method for people with voice trouble for which I had been searching for years. The influence on hyperfunctional voice troubles is usually not less rapid and effective than that on the voice of the deaf person. My experience includes many thousands of patients.

METHOD

The method should be used in the following way. The patient is asked to chew as usual with closed lips, but without anything in the mouth, and to observe his tongue, which moves continually during the chewing. Immediately afterward he is asked to chew "like a savage," that is, by opening the mouth, and with extensive movements of the lips and tongue. Many people, when chewing with open mouth, have the tongue continually on the floor of the mouth. But that, surely, is not the way savages may chew, who lift and drop the tip of the tongue. Only the original method of chewing guarantees the setting free of the voice. Then the patient must give voice during chewing. If the chewing is done correctly, that is, with vigorous movements of the lips and the tongue, a great variety of

sounds escape the mouth. If uniform sounds, like "ham-ham-ham," [11] are heard, the movements of the lips and tongue do not vary sufficiently. It is amazing how a hyperfunctional voice changes immediately into a free and normal one. Abnormally low female voices, as they sometimes occur with the change of voice (perverse change of voice), rise during chewing. That seems to be another proof of the theory that chewing with voice production is the original function of voice, and at the same time the origin of human speech. The natural female voice appears immediately during chewing! This fact also proves that the chewing method is not a "relaxation method," but a means of bringing forth the still present original association of voice and speech.

The patient should chew with voice production at least twenty times a day for a few seconds only, but should always avoid doing so mechanically. He should act with the guiding idea of having some food in his mouth. After a few days, he should read several times a day for one or two minutes only, but should begin with a voiced "nonsense" chewing and should always, after having read some words, introduce another "nonsense" chewing. It is explained to him that the different languages are nothing but chewing, and that his speech function is therefore fundamentally identical with the voiced chewing. In other words, he should maintain the idea of chewing also when he speaks a language.

The chewing method has, of course, nothing to do with swallowing, but may judiciously contain the idea of taking something in. In this way, it may help avoid the waste of breath which is a frequent trouble of singers and speakers, especially those with hyperfunction and hypofunction.

American readers of this paper may think that the use of chewing gum would help the patient get used to the chewing method. I oppose this. I prefer the idea of chewing to real chewing, for a person may forget the gum or may have no opportunity to use it, but he can always be sure of the presence of the idea in himself.

Nevertheless, gum chewing played an interesting part in my studies on the influence of chewing on voice and speech. Two years ago my wife and I made a trip on a Greyhound bus. The drivers changed regularly, and the new driver always addressed the travelers. I could then predict to my wife, without a single error, whether or not the driver chewed gum, making the differential diagnosis from the impression of a hyperfunctional or a hypofunctional voice, on the one hand, or of a normal voice, on the other. The normal voice indicated to me that the man was used to chewing gum.

It may be that the fact that American voices, especially those of men, are on the average better than those of Europeans is partly due to the exclusive use of chewing gum in the United States. Hollingsworth [12] found that muscular tension decreases during chewing. He examined subjects doing different kinds of work and noted a decrease in tension during chewing.

REPORT OF CASES

A few cases may illustrate the therapeutic procedure.

11. The formula "ham-ham-ham" is an age-old and widely used symbol to stimulate eating in small children. This symbol contains the vowel *a;* in other words, it is voiced. But the function of eating, which one wishes to stimulate, is not associated with voice production in the cultivated world. It may be that the vowel *a* comes into this symbol as a sign of subconscious remembering of the initial junction of chewing and voice production.

12. Hollingsworth, H. L.: Chewing as a Technique of Relaxation, Science **90**:385-387 (Oct. 27) 1939.

CASE 1.—A priest aged 67 had been suffering from voice trouble for many years. Thirty years ago he had been operated on for papillomas of the larynx. Sometimes during the last eight years he could hardly preach because his voice was hoarse and weak and his throat was "burning like fire." He felt he would have to resign his position. The voice was feeble, and there was a mixture of hyperfunctional and hypofunctional symptoms. No anatomic changes were noted in the larynx, but the pharynx was reddened and the veins of the palate were dilated. In a condition of this kind the hyperfunction should be treated first. Experience has proved that the signs of hypofunction frequently disappear if those of hyperfunction are relieved. I shall not describe here the treatment of hypofunction because the methods do not belong to the topic of hygiene of the voice. The first attempt to chew with voice brought about a normal voice during the chewing. After a daily treatment for three weeks, the priest went home, where he performs his duties with a sufficiently loud voice. Sometimes if he forgets the chewing, he feels trouble in his throat, which disappears if he resumes the therapy.

CASE 2.—A lawyer aged 48 caught cold three years ago, during a trial in which he worked for the defense. Before that time he had a powerful voice. For a number of months speaking presented great difficulties. The trouble diminished gradually, but it was still severe enough to hinder him in his profession. Laryngoscopic examination revealed no pathologic change. The pharynx was dark red, and the veins of the palate were dilated. The septum of the nose was slightly deviated. Hyperfunction of the strangulation type was present. The patient reacted promptly to treatment by the chewing method. The hyperfunction disappeared, but he wasted air in voiced chewing and speaking. He was, therefore, asked to think of taking in something during chewing. In this way the voice was restored perfectly in fifteen treatments, carried out over a period of five weeks.

CASE 3.—A politician aged 40 during the last three years had frequently had to speak in public. He sometimes became hoarse during a speech and had to exert great strength. After all the longer speeches his throat was very tired. Pain and paresthesias, which developed during speeches, did not disappear during rest of the voice. Because of the paresthesias, frequent hawking was necessary. He was treated locally by a laryngologist for weeks, with no improvement. Laryngoscopic examination showed slight redness of both vocal cords. The pharynx was dark red; the veins of the palate were enlarged. Hyperfunction of the *coup de glotte* and strangulation types were present. After a longer period of speaking, paretic hoarseness appeared. Treatment by the chewing method resulted in a normal and pleasing voice. The duration of therapy was ten weeks, with treatments three times a week.

CASE 4.—A rabbi aged 49 had hoarseness and steadily growing pains in the throat for ten or twelve years. The pharynx and larynx were treated locally, and rest of the voice was frequently ordered, but lasting improvement was no longer achieved in this way. Examination revealed slight redness of the vocal cords, hypertrophy of the middle right turbinate, reddening of the pharynx, extreme dilatation of the veins of the palate and signs of hyperfunction and hypofunction of the voice. A perfect cure with the chewing method was achieved in four weeks, with treatments four times a week.

CASE 5.—A woman aged 27, a professional singer, was a member of a highly musical family. She had often sung as a child as soloist in choruses. She did not know when the change in her voice took place. Systematic voice training began at the age of 17 and was interrupted after six months, when she married. She frequently sang arias and songs privately. Her throat always felt tired after a few songs. At the age of 22 she resumed voice training. The teacher advised her to locate every tone in the upper part of her face and to sing very loudly. After a year she changed to an Italian teacher, who trained especially for coloratura. Tonsillectomy was done twice during the period from 1918 to 1923, and the tonsils were treated with radium in 1928. On that occasion the laryngologist noted thickening of the vocal cords. Her first child was born in 1936. She stopped singing during the period of lactation. In 1938 she began work with a teacher who had studied with me in Vienna and used the chewing method. He found that she wasted air and that she pressed out the lateral parts of the throat in singing and in speaking. No hyperfunction was present after a training of six months. Her second childbirth was in 1940. She did no singing during the period of lactation. In January 1941 she had influenza for two weeks. Afterward the teacher noticed that "something was wrong with her voice" and sent her to a laryngologist, Dr. Franz Altmann. He observed a cyst the size of a kernel of hemp corn near the inner margin of the right vocal cord, in the typical place, and a small blood vessel extending from there toward the anterior commissure. The cyst was filled with a reddish brown fluid. He ordered complete rest of the voice during an observation period of two weeks. The cyst became a little smaller but did not change further in size in six weeks. She therefore was sent to me. In singing she wasted a moderate amount of air and there was slight hyperfunction of the strangulation type. But the speaking voice was accompanied by pronounced hyperfunction of the first and second

types and outward pressing of the lower lateral parts of the throat and swelling of the external jugular veins. I treated, therefore, only the speaking voice by the chewing method. After eight weeks the speaking voice was normal. The cyst was reduced to one-half its former size. Two months later the cyst disappeared, and there was only a thickening of the vocal cord the size of a pinpoint. The enlarged vessel did not change. The singing voice developed constantly. The patient became a very successful concert singer. Later, in 1941, tonsillectomy was performed by Dr. Altmann.

COMMENT

A method which has proved successful in cases of severe hyperfunctional and hypofunctional states of the voice and their sequelae is expected to be effective also in cases of less severe trouble. The method, indeed, has proved helpful in many hundreds of cases of hyperfunction of everyday life and also in cases of functional hoarseness in children. Likewise, in treatment of very severe breaks of the voice during the change at puberty, as well as in management of the pathologic sequelae of change of the voice, such as the eunuchoid voice and the "perverse voice change," the chewing method has been most successful.

The method, therefore, is one which can be used to avoid certain severe troubles of the voice, in other words, as a hygienic measure. It could easily be introduced into the kindergarten,[13] grammar school, high school and university. In the kindergarten, grammar school and high school, one minute of voice chewing at the beginning of every lesson, and a more extensive use of it in reading and speech lessons, would help to avoid, and to eliminate if necessary, hyperfunctions of the voice. In universities the method could be introduced in the courses of public speaking. After persons have finished school, they may use the chewing method by themselves, in professional groups or in groups especially formed for the purpose of this training. But any limited time, for instance, half an hour or an hour a day, for the practice of the method is not sufficient. Experience has shown that it is best to practice twenty times a day, each time for a few seconds only, but always with the guiding idea of the real chewing of an imaginary bite. The time which a person needs to make the muscles chew automatically in speaking cannot be predicted. It depends on the individual. Many persons never get entirely used to it, but they can learn to maintain the idea of chewing while they are thinking of what they are saying. But other methods are still less likely to become automatic, because they appeal to too many partially separate functions. Even if a person forgets the chewing method for a while, and relapses into his old habits, he can easily recall it and immediately correct his unhygienic movements.

Hygiene of the voice may even prevent organic disorders of the respiratory tract. The epithelium of the vocal cords, as well as that of the mucous linings of the throat, is injured by the continuous attack due to a hyperfunctional state[14] and therefore may be more susceptible to infection. The pressure of one vocal cord against the other in production of the *coup de glotte* may even favor a malignant growth in a predisposed person.

SUMMARY

The cries of the suckling are partly "hard," that is, hyperfunctional.

The hyperfunctional voice is associated with emotions, especially unpleasant ones. Six forms of hyperfunction, with the structures involved, are mentioned here.

13. Just now I am treating a boy of 4½ years, who easily understands and practices the chewing method.

14. F. Schmitz-Svevo showed that dryness of the throat and of the mouth disappears if the patient chews, or even thinks of chewing (Chewing Method Applied in Cases of Pharyngeal Dryness of Organic or Psychogenic Origin, J. Speech Disorders **6**:185-186 [Dec.] 1941).

Professional speakers and singers frequently suffer from "phonasthenia," which might be called a hyperfunctional or hypofunctional state with or without nervous sensations.

Many children, especially school children, show symptoms of hyperfunction, and a smaller number exhibit symptoms of hypofunction.

Real functional trouble of the voice always begins with hyperfunction.

The chewing method has proved a quickly effective, successful method for the treatment of hyperfunction of the vocal apparatus. It is therefore recommended also as a means of prevention of serious voice troubles.

The method could easily be introduced into kindergartens, grammar schools, high schools and universities, as well as into professional groups.

Hygiene of the voice is probably a means of diminishing the danger of infectious disease of the respiratory tract.

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RESPONSE OF THE LABYRINTHINE APPARATUS TO ELECTRICAL STIMULATION

SITE OF ACTION; FARADIC STIMULATION; INVERSE EFFECTS OF ANODIC AND CATHODIC STIMULATION

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Despite the enormous literature gathered since the classic work of Hitzig, Breuer and Ewald, the action of electrical stimuli on the labyrinthine apparatus [1] is not yet understood. The difficulties and contradictions encountered if one assumes that the current acts on the fibers or the peripheral endings of the vestibular nerve were summarized by Brünings.[2] Some of these difficulties are easily met; e. g., the continuation of nystagmus after the stimulation ceases is simply a phenomenon of after-discharge, which is encountered with many reflexes. The fact that the response not only appears on the closing and opening of the current, as on stimulation of motor nerves, but continues during the flow of the current is understandable if one bears in mind that electrical stimulation of sensory nerves follows other rules than those for motor nerves. Ebbecke,[3] for instance, demonstrated that stimulation of a sensory cutaneous nerve with a constant current of medium intensity produces a whizzing (vibrating) sensation (*Nervenschwirren*), lasting up to several minutes. In analogy with this phenomenon, Spiegel and Aronson [4] observed that the nystagmus elicited by galvanic stimulation shows a rhythmic waxing and waning of intensity.

Among the phenomena mentioned by Brünings, and recently also by Pollock, Finkelman and Sherman,[5] there are two in particular which are not adequately explained as yet: the ineffectiveness of faradic stimulation and the reversal of the motor responses on reversal of the direction of the stimulating direct current. An analysis of these phenomena will be attempted in the present paper. Before we approach these problems, it seems advisable to present data regarding the site of action of the galvanic stimulation.

SITE OF ACTION OF GALVANIC STIMULATION

Three possibilities of the site of action of the galvanic current should be considered: (1) the peripheral sense organ; (2) the peripheral sensory neuron of

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1. The general term "labyrinthine apparatus" is chosen because the site of action is under discussion.

2. Brünings, W.: Verhandl. d. deutsch. otol. Gesellsch. **19**:192, 1910.

3. Ebbecke, U.: Arch. f. d. ges. Physiol. **197**:482, 1923.

4. Spiegel, E., and Aronson, L.: Continuous Stimulation of Labyrinth with Sustained Nystagmus, Arch. Otolaryng. **17**:311 (March) 1933.

5. Pollock, L.; Finkelman, I., and Sherman, I. C.: J. Nerv. & Ment. Dis. **93**:473, 1941.

the vestibular nerve and/or (3) central parts of the vestibular pathways (vestibular nuclei and their fibers).

In agreement with the results of Neumann [6] and Marx,[7] we observed that elimination of the peripheral sense organ, e. g., by mechanical destruction of the inner ear, does not prevent the appearance of typical responses on application of a galvanic current, and that the slow component of the responses (deviation of the eyeballs and rotation of the head) is still directed toward the anode and the quick component toward the cathode.

A comparison of the threshold values before and after the labyrinthectomy sometimes even revealed lower values after this operation (e. g., in rabbit 11, with electrodes placed in the external meatus, nystagmus was produced before operation at 8 to 15 milliamperes and afterward at 3 to 5 milliamperes). This paradoxic result probably was due to the fact that the cavity produced by the labyrinthectomy facilitated the conduction of the current to the vestibular nerve.

Thus, the current acts central to the peripheral sense organ, on the peripheral neuron of the vestibular nerve or on the vestibular nuclei and their fiber systems. There exist contradictory statements in this respect. In experiments of Dohlman [8] on rabbits intracranial section of the eighth nerve abolished or prevented the reactions to galvanic stimulation, while Huizinga [9] still obtained such reactions in pigeons after extirpation of the ganglion vestibulare Scarpae. Huizinga [9] explained Dohlman's results as due to shock and the poor general condition of his animals.

In order to obviate the objection of shock, we performed the experiments on decerebrate cats, so that we dealt with preparations the reflex excitability of which was decidedly increased. After decerebration one cannot, of course, expect to obtain ocular reactions, since the transection of the midbrain as a rule cuts also the nerves of the ocular muscles at the base of the brain. The tonic rotation of the head about the oro-occipital axis toward the anode is, however, pronounced in decerebrate cats and may be used as a good indicator of the excitation of the labyrinthine apparatus by a direct galvanic current flowing transversely between the ears. On monaural stimulation by means of a bipolar electrode inserted into the round window or into the inner ear after the promontory has been chiseled off, one obtains in such decerebrate cats a rotation of the head toward the nonstimulated ear. Unilateral intracranial section of the eighth nerve on the side of stimulation prevented the rotation to the opposite side produced by monaural bipolar stimulation. Bilateral section of the eighth nerve abolished the rotation toward the anode that normally is elicited by a direct current flowing between the two ears.

The loss of these reactions following the intracranial section of the eighth nerve could not be ascribed to the poor condition of the preparation or to "shock," since the current still elicited reactions after elimination of the eighth nerves. There appeared now, on monaural bipolar stimulation, rotation of the head to the same side and, on binaural stimulation, rotation to the cathode side, reactions which are due to reflex stimulation of afferent fibers in the fifth and the ninth and tenth nerves, respectively, as was shown previously by one of us (Spiegel [10]).

In a second series of experiments, the effect of intracranial section of the eighth nerve was tested in cats which were not subjected to decerebration by transection of the midbrain, but in which the cerebral hemispheres were ablated while the

6. Neumann, H., in discussion on Brünings.[2]
7. Marx, H.: Ztschr. f. Ohrenh. **63**:201, 1911.
8. Dohlman, G.: Acta oto-laryng., 1929, supp. 8, p. 1.
9. Huizinga, E.: Arch. f. d. ges. Physiol. **226**:709, 1931.
10. Spiegel, E.: Am. J. Physiol. **135**:628, 1942.

base of the brain and the nerves to the ocular muscles were spared. The intactness of the efferent innervation of the ocular muscles was indicated by the fact that illumination still produced miosis of the pupils; in other words, the centrifugal fibers to the sphincter of the pupil, which are rather sensitive to injury, still were able to conduct reflex impulses to the periphery. After bilateral intracranial section of the eighth nerves, currents up to 15 milliamperes flowing transversely between the ears were unable to elicit ocular movements, although such currents produced dilatation of the pupils and general defense reactions and normally much smaller currents (2 to 4 milliamperes) were sufficient to produce nystagmus. Thus, in agreement with Dohlman, no evidence could be obtained that the reactions normally produced by monaural or by binaural stimulation are caused by an action of the current on the vestibular nuclei or their fiber connections with the nuclei of the motor nerves to the ocular muscles. The different results of Huizinga may be due either to the fact that conditions are different in pigeons or to incomplete extirpation of Scarpa's ganglion. The latter interpretation is suggested by the fact that Huizinga observed disappearance of the galvanic reaction in pigeons in which he had extirpated the labyrinth if some months had elapsed after the operation,[11] so that secondary degeneration had developed in Scarpa's ganglion. Blonder and Davis [12] also observed that section of the eighth nerves abolished reactions in the extremities elicited by galvanic stimulation of the vestibular apparatus.

In summing up, we arrive at the conclusion that on electrical stimulation of the vestibular apparatus (monaural or binaural stimulation) a typical response is still obtained after destruction of the peripheral sense organ, but is no longer elicited after intracranial section of the eighth nerves. Thus, the current acts on the peripheral neuron of the vestibular nerve (or parts of this neuron).

FARADIC STIMULATION

The conclusion arrived at in the preceding paragraph seems to be incompatible with the experience cited in all handbooks and textbooks that faradic stimulation is unable to excite the labyrinthine apparatus, no instance being known in which a motor or a sensory nerve would not respond to faradic stimulation.

A first step in an approach to this problem was made in a former study by one of us (Spiegel [13]); it was ascertained whether the labyrinth is able to respond to a rhythmically interrupted direct current. In experiments on cats in which a bipolar needle electrode was introduced into the inner ear, it was found that nystagmic jerks with a slow and a fast component could be elicited by a current interrupted at frequencies below 10 per second. If the frequency of the current was increased, the eyes showed oscillations without a slow and a fast component, and eventually a tremor, the amplitude of which diminished with increasing frequency. With frequencies of stimulation above 27 per second definite oscillations could no longer be recorded. It was first thought that this phenomenon might be related to the rather long chronaxia (14 to 22 milliseconds) reported by

11. In mammals (guinea pigs), also, several months after labyrinthectomy unexcitability of the vestibular apparatus may develop (Brünings, W.: Ztschr. f. Ohrenh. **63**:20, 1911), apparently as a result of the development of degenerative changes in the peripheral neuron of the vestibular nerve.

12. Blonder, E. J., and Davis, L.: The Galvanic Falling Reaction in Patients with Verified Intracranial Neoplasms, J. A. M. A. **107**:411 (Aug. 8) 1936.

13. Spiegel, E. A.: Rhythmic Stimulation of the Labyrinth, Arch. Otolaryng. **33**:572 (April) 1941.

Bourguignon and Dejeau [14] for the vestibular nerve. However, the values obtained by one of us (Spiegel [13]) were much lower than theirs.

In these experiments a rotating interrupter similar to Boruttau's [15] chronaximeter was used. This permitted one to apply a pulsating unidirectional current with variable duration of the single stimuli. In order to observe the effect of the vestibular stimulation undisturbed by volitional movements, the measurements were made on decerebrate cats. In these preparations a reaction similar to the inclination of the head toward the anode used by Bourguignon and Dejeau with human subjects can easily be elicited, namely, the aforementioned rotation of the head about the oro-occipital axis toward the anode on binaural stimulation, or toward the nonstimulated ear on bipolar monaural stimulation. These reactions could still be elicited by the pulsating unidirectional current at a rheobase of 20 to 40 volts and with a frequency of 55 stimuli per second, if the duration of the single stimuli was lowered as far as to 0.9 to 1.8 milliseconds. These values are close to those reported by Dohlman and Betsholtz [16] (1 to 2 milliseconds) for deviation of the eyeballs in human subjects. Pollock, Finkelman and Sherman [5] observed past pointing even with stimuli of a duration of 0.03 millisecond at 1,500 stimuli per second and 12.4 milliamperes (voltage not given).

Bipolar monaural stimulation of the inner ear with rectangular alternating current also elicited the rotation of the head to the opposite side if the duration of the single phase was reduced to 1.8 milliseconds (at 20 volts). Occasionally, only an alternating current with a duration of the single phase above 2.7 milliseconds was able to produce this reaction.

Thus, the inability of the vestibulo-ocular reflex arc to respond by distinct rhythmic ocular jerks to a current interrupted at frequencies above 27 per second could not be explained by the chronaxia of the vestibular nerve. This value, rather, suggested that the vestibular nerve should be able to respond to currents of higher frequencies; it seemed advisable, therefore, to reexamine the problem of the alleged unexcitability of the vestibular nerve by faradic current and to use as an index, in a manner similar to that employed in the measurements of chronaxia, the rotation of the head instead of the ocular reactions.

These experiments were again performed on decerebrate cats in which a bipolar electrode, usually of the concentric needle type, was inserted into the round window or into a cavity of the inner ear. A special clamp fixed on the head holder kept the electrode rigidly in this position, independent of movements of the head. The head holder (Czermak type) was either loosely held by the examiner, so that movements of the head in all directions were possible, or placed with its handle in a sling so that the head could freely rotate about the oro-occipital axis.

With this arrangement it could easily be demonstrated that bipolar monaural stimulation by a direct current produced a rotation of the head about the oro-occipital axis toward the nonstimulated ear so that, as a result of this reaction, the nonstimulated ear lay lower than the stimulated one (contralateral cephalogyric reaction). The same reaction could be obtained by stimulation with faradic current, a Harvard inductorium at a coil distance of from 11 to 13 cm. and with 4 volts in the circuit of the primary coil being used.

The reaction may be associated with retraction of the head and/or turning of the head about a dorsoventral axis toward the opposite shoulder and extension

14. Bourguignon, G., and Dejeau: Rev. neurol. **34**:1017, 1927; Compt. rend. Soc. de biol. **101**:1057, 1929.

15. Boruttau, H.: Ztschr. f. d. ges. Neurol. u. Psychiat. **83**:284, 1923.

16. Dohlman, G., and Betsholtz, T., abstracted, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **23**: 58, 1934.

of the homolateral foreleg. This extension of the homolateral foreleg may elevate the corresponding shoulder and thus produce an inclination of the head that may increase or simulate the contralateral cephalogyric reaction. It should, however, be emphasized that the rotation of the head toward the opposite ear could also be observed independently of this reaction of the foreleg. It is also important to note that this contralateral cephalogyric reaction on bipolar monaural stimulation with faradic current could only be elicited if the electrodes were inserted into the round window or into a cavity of the inner ear, while faradic stimulation of the external or the middle ear produced only retraction of the head and/or rotation in an irregular direction. Apparently, the pure effect of vestibular stimulation can be elicited by faradic stimulation only if the bipolar electrodes are applied as closely as possible to the eighth nerves. Otherwise, the current easily spreads to the neighboring afferent nerve roots (fifth, ninth and tenth), and the effect on the eighth nerve is masked by reflex effects from these fibers, such as rotation of the head to the same side. This may also be the reason that faradic stimulation in man with the stimulating electrode applied to the external ear fails to elicit the typical vestibular tonic reactions.[17]

On repeated examination the contralateral cephalogyric reaction may become exhausted and be also replaced by rotation of the head toward the stimulated side. This homolateral cephalogyric reaction is of a nonlabyrinthine origin, as was previously shown by one of us (Spiegel [10]).

In order to ascertain whether the contralateral cephalogyric reaction appearing on faradic stimulation of the inner ear is due to excitation of the peripheral neuron of the vestibular nerve, the effect of intracranial section of the eighth nerve on this reaction was tested. This operation abolished or prevented the contralateral cephalogyric reaction, which was replaced by the rotation to the same side. The associated retraction of the head and the turning about a dorsoventral axis toward the opposite shoulder could still be elicited after section of the eighth nerve; these reactions, therefore, are at least partly nonlabyrinthine in origin. In control experiments, the roots of the afferent nerves adjoining the vestibular nerve, i. e., the fifth, ninth and tenth nerve roots, were cut on the side of the stimulation, while the eighth nerve was left intact. This procedure did not prevent the contralateral cephalogyric reaction on faradic stimulation of the inner ear.

Thus, a reflex response to faradic stimulation of the vestibular nerve could be obtained, and the doctrine of unexcitability of this nerve by faradic current can no longer be maintained.

The experience that faradic current is able to elicit a vestibular response, such as the contralateral cephalogyric reaction, but is unable to produce nystagmus requires an explanation, which may perhaps be sought in the following direction. It has been seen that rhythmically interrupted direct current was able to elicit nystagmic movements of the eyes only at a rather low frequency, that with increasing rate of the stimuli only a tremor of diminishing amplitude could be elicited and that with stimuli above 27 per second definite oscillations of the eyeballs could no longer be recorded. A similar phenomenon could be observed on stimulation with rectangular alternating current if the rate of the phases per second was increased. The inability of faradic current to elicit nystagmic movements of the eyes seems to be related to this phenomenon. One has to bear in mind that nystagmus represents alternating reactions of the antagonistic dextrogyric and levogyric neuromuscular apparatus of the eyeballs. The experience with

17. On binaural application of the electrodes one can, of course, hardly expect that an alternating current would produce tonic reactions directed to the left or the right side.

rhythmically interrupted direct current just described seems to indicate that the neuromuscular mechanism of these alternating reactions has a certain inertia, being unable to follow rhythmic impulses from the peripheral nerve above a certain frequency, while a one-phasic reaction, such as the rotation of the head, may be elicited by rhythmic stimuli of much higher frequency.

INVERSE EFFECTS OF ANODIC AND CATHODIC STIMULATION

It has been rather puzzling to all observers that the direction of the motor responses to galvanic stimulation of the labyrinthine apparatus depends on the direction of the current, the deviation of the eyeballs and the slow component of nystagmus, as well as the deviation of the head and the falling reaction being directed toward the anode and away from the cathode.

Explanations that assume an action of the current on the central nervous system or on the peripheral sense organ need no further detailed discussion, since an action of the current on these parts of the reflex arc is refuted in the section on "Site of Action of Galvanic Stimulation." The possibility may be considered that, with the labyrinth intact, an electrokinetic (catophorectic or electro-osmotic) action of the current on the endolymph, as proposed by Brünings [2] and by Pollock, Finkelman and Sherman,[5] may be a supporting factor.

Observations on the cupula of the living pike (Steinhausen [18]), however, showed that only stimulation of the labyrinth by rotation or by changes of temperature produces a flow in endolymph resulting in deviation of the cupula, while the typical ocular reactions on galvanic stimulation appear without deviation of the cupula. Thus, the problem is reduced to the question why opposite effects should be brought about by cathodic and by anodic stimulation of the peripheral neuron of the vestibular nerve. This phenomenon could be due to quantitative or to qualitative differences in the action of the two poles (or of the different directions of the current). The first possibility was considered by Wilson and Pike.[19] They assumed that the vestibular nerve contains two sets of fibers and that the endings of the one set producing deviation of the eyeballs to the same side are more easily excited but less numerous than those of the other set, which produce ocular deviation to the opposite side. The stronger stimulus, i. e., the cathodic, will bring about excitation of the more numerous fibers and will produce deviation to the opposite side; the weaker stimulus, the anodic, will act on the more excitable, less numerous fibers and produce deviation to the same side. Thus, this theory explains the opposite effects of anodic and cathodic stimulation by quantitative differences in the action of the two poles and in the excitability of the supposed two sets of fibers. According to this conception, one should expect that on application of a gradually increasing current one and the same pole, e. g., the cathode, should first produce the effects of the weaker anode, and on further increase of the current the opposite, cathodic effect.

We tried, therefore, to ascertain whether monopolar cathodic stimulation would produce opposite effects if the intensity of the stimulating current was gradually increased. The experiments were again performed on cats. The stimulating electrode was inserted either into the external meatus or into a cavity of the inner ear; the diffuse electrode was placed on the shaved abdomen of the animal. In all experiments the typical cathodic effect was observed when the threshold was

18. Steinhausen, W.: Arch. f. d. ges. Physiol. **232**:500, 1933.
19. Wilson, J. G., and Pike, F. H.: Phil. Tr. Roy. Soc., London, s.B **203**:127, 1912.

reached (e. g., at 1 to 2 milliamperes, rolling of the eyeballs to the opposite side; at 3 to 4 milliamperes, nystagmus to the same side), but the appearance of the cathodic response was never preceded by a reaction in the reverse direction.

Qualitative differences between the anodic and the cathodic effects were assumed by Bárány in that the cathode should act by increasing, and the anode by depressing, the excitability and conductivity of the vestibular nerve. It seems hard, however, to demonstrate such a depressor action of the anode. Brunner,[20] for instance, combining binaural application of the anode and unilateral calorization, failed to find a depressor influence of the anode.

In approaching this problem, we tried to ascertain whether binaural anodic stimulation would influence the excitability of the labyrinths to rotation. In each external meatus an electrode was inserted, and both were connected with the anode, while the cathode was connected to a diffuse electrode fastened to the abdomen of the cat. Special care was taken to insert both ear electrodes in the same way, so that the current entered the inner ear on both sides under as equal conditions as possible. The animals were tested by ten rotations to the right and to the left without and with application of a direct current. The duration and number of jerks of the postrotatory nystagmus were determined. Currents up to 18 milliamperes were used and were applied during the rotation and the appearance of the postrotatory nystagmus. Significant differences between the values obtained on rotation without application of the direct current and those obtained on rotation accompanied by the flow of galvanic current could not be found (e. g., postrotatory nystagmus without application of the current, 10 to 25 jerks in eleven to nineteen seconds; postrotatory nystagmus on binaural application of the anode during and after rotation, 9 to 22 jerks in eleven to fifteen seconds).

The question arises whether these results refute the concept of a depressor effect of the anode. One must bear in mind that the experiments described deal with the effect of the anodic stimulation on the excitability of the semicircular canals only. The galvanic stimulation, however, chiefly affects fibers from the maculas, as shown by the fact that the primary reaction is a rolling of the eyeballs (Struycken[21]) and an inclination of the head. Thus, it seemed necessary to ascertain how anodic and cathodic stimulation, respectively, affect the continuous (tonic) impulses from the labyrinth. Decerebrate cats are excellent objects for such a study. The effect of monaural stimulation is rather equivocal for our problem, since the homolateral and the contralateral extremities are inversely influenced (Blonder and Davis[12]). More equivocal results may, however, be obtained if one stimulates both ears simultaneously.

A stimulating electrode was placed into each external meatus, and both electrodes were connected to the positive or to the negative pole, while the diffuse electrode, applied to the abdomen, was connected to the other pole. With such an arrangement, it could be demonstrated on decerebrate cats that bilateral cathodic stimulation increased the extensor rigidity of both forelegs; it could also be demonstrated that the extensor tonus, if well developed, was lowered bilaterally by anodic stimulation.

Thus, anodic stimulation, while unable to lower the excitability of the receptors in the semicircular canals, depresses tonic impulses sent by the labyrinth to the central nervous system. It may perhaps be assumed, as a working hypothesis, that this limited depressor action of the anode on some tonic labyrinthine impulses

20. Brunner, H., cited by Junger, J.: Monatschr. f. Ohrenh. **56**:451, 1922.
21. Struycken, H.: Ztschr. f. Hals-, Nasen- u. Ohrenh. **12**:627, 1925.

produces an imbalance between the vestibular nuclei of the left and the right side, sufficient to induce the nystagmus with the quick component in a direction away from this depressor effect.

SUMMARY

1. On electrical stimulation of the vestibular apparatus (monaural or binaural stimulation) typical responses were still obtained after destruction of the peripheral sense organ, while intracranial section of the eighth nerves prevented such reactions. It is concluded that the electrical stimulus acts on the peripheral neuron of the vestibular nerve or parts of this neuron.

2. Faradic stimulation of the vestibular nerve, while unable to elicit definite nystagmus, could produce a one-phasic reflex response, such as rotation of the head to the opposite side (contralateral cephalogyric reaction). Thus, the doctrine of unexcitability of the vestibular nerve by faradic current cannot be maintained.

3. The chronaxia of the vestibular nerve was determined, the contralateral cephalogyric reaction serving as criterion (liminal response). Values of from 0.9 to 1.8 milliseconds were obtained.

4. In an attempt to ascertain the mechanism of the inverse effects of anodic and cathodic stimulation, no evidence could be obtained indicating that this phenomenon is due to quantitative differences in the action of the two poles on two antagonistic sets of vestibular fibers. A depressor action of the anode on the receptors of the semicircular canals could also be excluded. However, a depressor action of the anode limited to some tonic labyrinthine impulses could be found, which may be responsible for the inverse effects of anodic and cathodic stimulation.

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BONE FISTULA

FURTHER STUDIES

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Much emphasis has been placed on the importance of laying epithelium over the fistula, particularly the epithelium of Shrapnell's membrane. We have reviewed the international literature on the effects of epitheliums on osteogenesis and nowhere find any evidence of an inhibitory effect. On the contrary certain epitheliums, notably of the genitourinary tract, stimulate osteogenesis.[1] We have reviewed all available literature on the effects of metals, heat, irradiation, presence of bone ash and other factors on osteogenesis in bone defects. It is our impression that the evidence does not support the claims advanced as to the efficacy of any of these factors in influencing osteogenesis favorably or otherwise in a manner sufficiently clear-cut to provide an adequate physiologic basis for an operation to create a fistula. That the operation is successful in some cases is an established fact. But the factors influencing the outcome are too ill understood to insure predictability.

In the past some statements have appeared in otologic literature touching on osteogenesis in artificial fistulas and on the various factors influencing it which are not supported by experimental evidence or by a correct interpretation of the relevant facts of the physiology of bone. The repeatedly voiced objection to the use of the rabbit cranium for experimental research with reference to this operation is an example.[2] This objection is based on confusion of two separate considerations: (1) the purely scientific problem of preventing osteogenesis in an artificial fistula of bone and (2) the application of the results of the study of this problem to the labyrinthine capsule. The problem in its present stage, we believe, is purely one of the physiology of bone and can be investigated on any convenient bone; for, as we have pointed out,[3] healing of defects in bone, whether in membrane or in cartilage bone, is normally a membrane bone process, whereas cartilage bone formation is an ontogenetic occurrence, never a repair process. Therefore we do not hesitate to trust physiologic principles demonstrated on a rabbit's cranium when discussing the behavior of labyrinthine fistulas, but we do ask

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1. Iselin, M.: Bone Development in Scar of Cystostomy, Bull. et mém. Soc. de chir. de Paris **20**:152 (Feb. 27) 1928. Shohl, A. T.: Mineral Metabolism, American Chemical Society Monograph Series, no. 82, New York, Reinhold Publishing Corporation, 1939. Leriche, R., and Lucinesco, E.: Heterotopic Osteogenesis Obtained by Grafting Strips of Vesical Mucosa into Muscle or by Grafting Aponeurosis into Bladder: Experimental Study, Presse méd. **43**: 137 (Jan. 26) 1935. Severi, R.: Question of Whether Heterotopic Ossification of Transplanted Urinary Mucosa Is Due to Mucosal Elaboration of Phosphatase of Osseous Type, Boll. Soc. ital. biol. sper. **14**:456 (Aug.-Sept.) 1939. Huggins, C. B.; McCarroll, H. R., and Blocksom, B. H., Jr.: Experiments on the Theory of Osteogenesis, Arch. Surg. **32**:915 (June) 1936. Tosatti, E.: Effects of Transplants from Renal Pelvis on Healing of Defects of Bones, J. Internat. Coll. Surgeons **3**:126 (April) 1940.

2. Kopetzky, S. V.: Operations on Labyrinthine Capsule for Otosclerosis, Surg., Gynec. & Obst. **72**:473 (Feb. 15) 1941.

3. Guggenheim, L. K., and Guggenheim, P.: Artificial Fistula in Cases of Otosclerosis, Arch. Otolaryng. **32**:1 (July) 1942.

the question: Will the minute and delicate structures of the labyrinth withstand the procedure necessary to produce a patent fistula? The latter is a separate consideration for the future, of interest to the surgeon, but in no way relevant to the fundamental physiologic question with which we are at present concerned.

It occurred to one of us (P. G.) to approach the problem of the fistula from a biochemical point of view inasmuch as no use had (to our knowledge) been made of the available facts of the chemistry of bone formation. With the idea of interfering in a specific and selective way with the chain of chemical events leading to ossification, we constructed on theoretic grounds a list of chemical agents and submitted them to analysis with respect to practicability. The agents were copper, silver, an iodoacetate, a fluoride and strontium.

Copper ions, even in minute numbers, are known to catalyze the oxidation of vitamin C. In the absence of vitamin C, collagen cannot gel, and therefore the first step in the formation of the matrix of bone would be prevented.

Silver ions are known to inactivate phosphatase, thereby possibly inhibiting the calcification of gelled collagen.

An iodoacetate is a "carbohydrate poison," interfering with the carbohydrate cycle by blocking the reaction: dioxyacetone phosphoric acid + glycerialdehyde phosphoric acid → phosphoglyceric acid + glycerophosphate. Since the carbohydrate cycle is probably the ultimate source of energy for all physiologic processes, it is conceivable that interference with it might lead to a disturbance of osteogenesis.

A fluoride is also a "carbohydrate poison," interfering with the transformation of beta phosphoglyceric acid to phosphopyruvic acid.

Strontium ions are known to be capable of producing rickets by impairing calcification.

At the time this analysis was made we had seen no mention in the literature of copper as an inhibiter of osteogenesis. We have since discovered through the work of Engström [4] that this matter has been given attention by French physiologists. The subject will be discussed later. Laszt and Verzar [5] reported osteoporosis in young rats from feeding sodium iodoacetate, but their work has been seriously questioned. Glock [6] reported that sodium fluoride in low concentrations inhibited the acid phosphatases of liver, kidney and erythrocytes in vitro but not the alkaline phosphatases of bone, serum or kidney unless these were in an acid medium. He also reported a temporary fluoride inhibition of osteogenesis in the early period of growth by virtue of a diminution of glycogen in the cartilage cells, the presence of which is important in calcification (carbohydrate vis a tergo?). Sobel, Cohen and Kramer [7] discussed the mechanism of the inhibition of calcification in the rickets induced with strontium and concluded that strontium does not inhibit the hydrolyzing effect of phosphatases. Like magnesium, however, it can form a nonionized complex with phosphate ions, or even a nonionized strontium-calcium phosphate complex. Competition of calcium and strontium ions for some factor in the bone cell or diminution by strontium of some uknown factor in calcification were mentioned as theoretic possibilities. Guggenheim, Gunther, Goodhill and Irvine [8] accomplished decalcification of the aural capsule in the white rat with parathyroid extract, viosterol and ammonium chloride. Ammonium chloride

4. Engström, H.: Ueber das Vorkommen der Otosklerose nebst eperimentellen Studien über chirurgische Behandlung der Krankheit, Acta oto-laryng., 1940, supp. 43, pp. 1-153.

5. Laszt, L., and Verzár, F.: Hemmung des Wachstums durch Jodessigsäure und antagonistische Beinflussung durch Vitamin B_2 sowie Nebennierenrinden-Hormon, Arch. f. d. ges. Physiol. **236**:693, 1935.

6. Glock, G. C.: Glycogen and Calcification, J. Physiol. **98**:1 (March 14) 1940.

7. Sobel, A. E.; Cohen, J., and Kramer, B.: The Nature of the Injury to the Calcification Mechanism in Rickets Due to Strontium, Biochem. J. **29**:2640 (Dec.) 1935; Phosphatase Activity and Calcification in Strontium Rickets, ibid. **29**:2646 (Dec.) 1935.

8. Guggenheim, L.; Gunther, L.; Goodhill, V., and Irvine, M.: Reversal of Halisteresis in the Mammalion Ear, Arch. Otolaryng. **34**:501 (Sept.) 1941.

produced the most selective and marked effect on the aural capsule, and the process was shown to be reversible when ammonium chloride was withheld and the rat placed on a high calcium diet and adequate vitamins. The possibility of accomplishing this was merely theory until this experimental basis was forthcoming to show that diet and vitamins did affect the state of calcification of the aural capsule. The implications for otosclerosis are clear from an elementary consideration of the histogenesis of the otosclerotic focus and are borne out by abundant clinical experience to be published in the near future.

There are two ways in which chemical agents could conceivably be brought to bear on the repair processes in a fistula of bone: local application and systemic administration. Aside from the inadvisability of attempting to inhibit so minute a bone process by upsetting the general metabolism, any systemic depression of calcium metabolism (such as Kend [9] is reported to be carrying out with diet and vitamins) would exert a pernicious effect on the otosclerotic process itself, increasing the halisteresis surrounding the focus of dystrophic bone and thereby inducing more rapid spread of the focus. We have therefore restricted our investigations to topical applications of chemicals to the area of the fistula. Here there arises the difficulty of maintaining for a sufficient time an adequate local concentration of the various agents, all of which are crystalloids. It seems probable that the local concentrations would drop almost to nil in a short time even were the fistula to be packed with dry powder or crystals.[10] It was therefore necessary to discard those agents depending for their action on a large local concentration of ions (mass effect), viz., the iodoacetate, strontium and the fluoride. Silver and copper remain. It is unlikely that the inevitable reduction of silver ions to metallic silver by the tissues would leave a sufficient concentration of ions to have any lasting specific effect on phosphatase. As previously stated, we have recently discovered that the idea of inhibiting osteogenesis chemically is not original with us. Engström,[4] who applied thin leaves of copper, silver and gold to the region of the fistula, stated that they hinder to a certain degree the new formation of bone in the following order of efficiency, according to the rule of Menegaux and Odiette: Cu > Ag > Au. Gold showed practically no activity. Copper produced an intense cellular reaction, resulting in dense fibrosis of the fistula. The effects were believed to be partly chemical, partly mechanical. Some of the experiments were carried out on labyrinths; others, on tibias. Menegaux and Odiette, whose investigations of the effects of metals on bone are apparently well known to continental otologists (although there is no mention of them in the Anglo-Saxon literature), seem to have been primarily interested in establishing the use of various metals and alloys in orthopedic surgery on a physiologic basis. Disks of various metals [11] were dropped into tissue cultures of osteoblasts obtained from 9 day chick embryos. It was found that magnesium and copper produced signs of cellular degeneration. Silver proved less harmful. The results were interpreted as partly indirect, brought about by changes induced in the mediums (e. g., increased alkalinity in the case of magnesium), and partly direct, due to toxic action of metal salts on the cells. These studies were repeated with the same results on human osteoblasts cultivated in vitro.[12] Studies were made of the effects of metals

9. Kend, L., cited by Kopetzky.[2]

10. Borsook, H.: Personal communication to the author.

11. Ménégaux, G.; Odiette, D., and Moÿse, P.: Action cytotoxique de quelques métaux sur le tissu osseux cultivé "en vie ralentie" ; contribution à l'étude biologique de l'ostéogsynthèse, Presse méd. **42**:1844 (Nov. 17) 1934.

12. Ménégaux, G., and Odiette, D.: Action cytotoxique de certains métaux des ostéoblasts humains cultivés "in vitro," Presse méd. **43**:1555 (Oct. 5) 1935.

on the deposition of calcium in tissue cultures of osteoblasts by the microincineration technic of Policard.[18] Aluminum, which produced no degenerative changes in the cells, was found to inhibit strongly the fixation of calcium. Iron also was inhibitory but, like magnesium, prevented the growth of osteoblasts. The authors did not speculate as to the actual chemical mechanisms of these effects, speaking merely of the "cytotoxic action" of the metals.

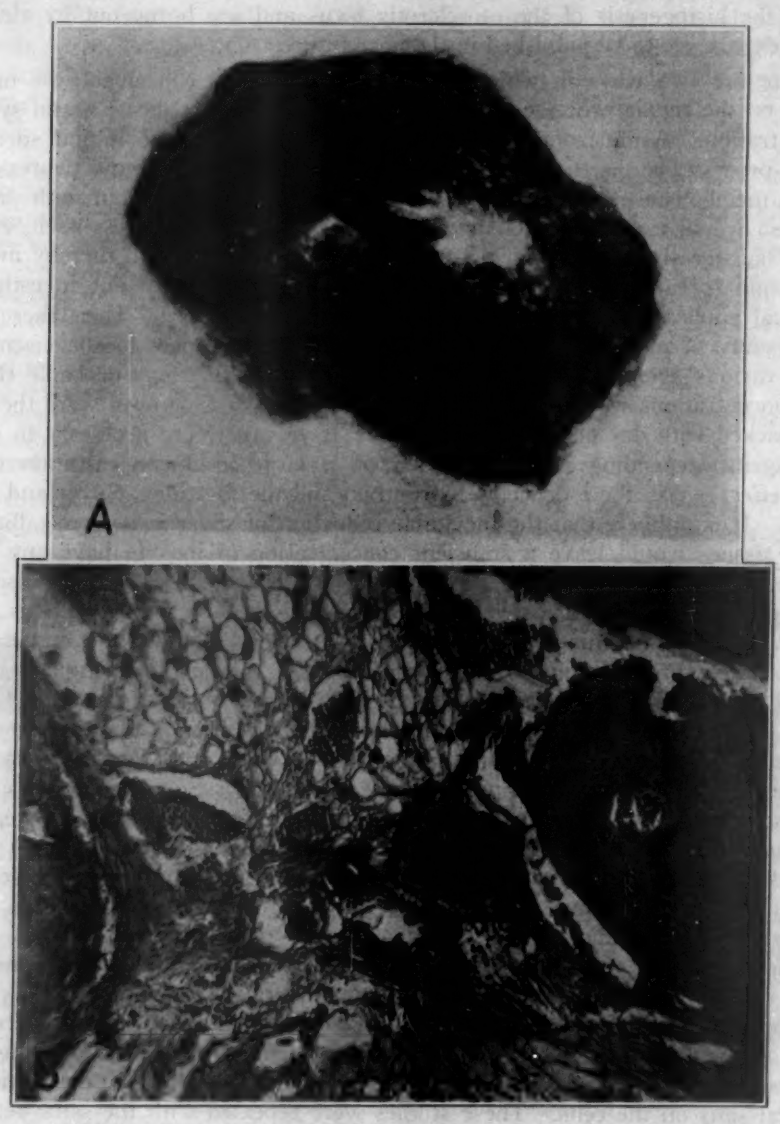

Fig. 1 (experiment 1).—*A*, roentgenogram of an osseous fistula treated with saturated solution of copper sulfate and copper wire. *B*, photomicrograph of the content of the fistula after two months and eleven days.

The experiments which we have carried out differ from the aforementioned ones in that *solutions* of the metal salts were applied to bone fistulas. Copper

13. Ménégaux, G., and Odiette, D.: Influence du quelques métaux sur la fixation des composés minéraux dons les cultures d'osteoblasts, Presse méd. **43**:152 (Jan. 26) 1935.

was believed to hold the only promise and was applied in the form of copper sulfate solution in three different strengths and in the form of wire. Silver nitrate and trichloroacetic acid were used as controls in an attempt to demonstrate that the action of copper was, in fact, due to a specific chemical mechanism of the type discussed and not to any nonspecific caustic or other action.

EXPERIMENTS

General Procedure.—In all of the experiments adult white rabbits were used. Operations were done on the cranium with the animal under anesthesia induced with ether. No attempt was made to suture the periosteum after completion of the procedures, the skin simply being closed with catgut. At the termination of each experiment the areas of the fistulas were removed and roentgenographic studies made before the specimens were decalcified for sectioning.

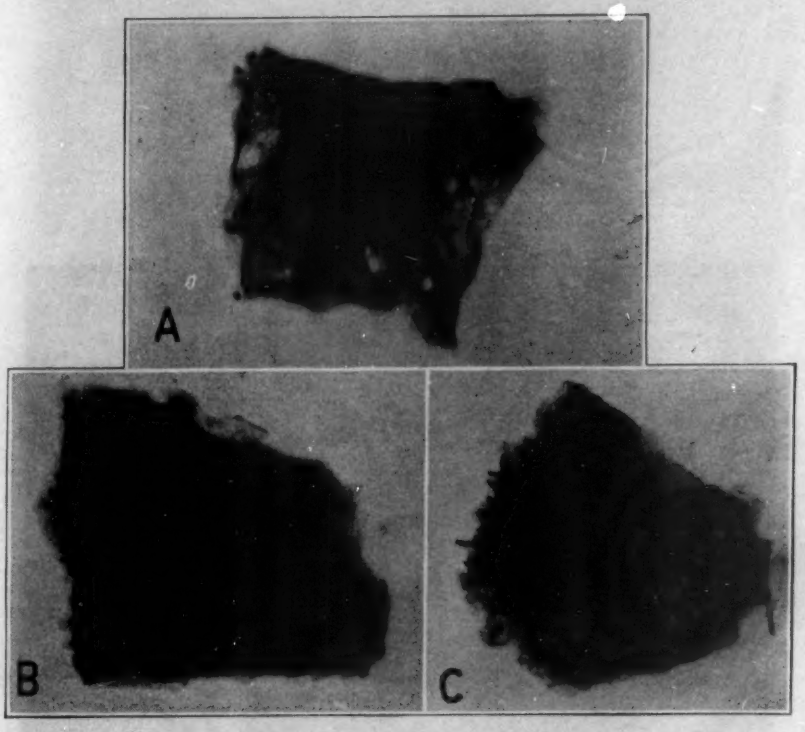

Fig. 2.—*A* (experiment 2), roentgenogram of an osseous fistula treated with saturated solution of copper sulfate (complete closure). *B* (experiment 3), roentgenogram of an osseous fistula treated with copper wire (complete closure). *C* (experiment 4), roentgenogram of an osseous fistula treated with a 1:10 dilution of a saturated copper sulfate solution (complete closure).

Experiment 1.—Procedure: On Feb. 12, 1941 an osseous fistula was made with a sharp chisel, and over a ledge of bone projecting into it a piece of fine copper wire was tied. The entire fistula was painted with saturated copper sulfate solution. On April 23 (two months and eleven days later) the region of the fistula was removed. A preliminary report of this experiment has been published.[14]

Result: The roentgenogram (fig. 1*A*) shows a patent fistula. On one wall is the projecting ledge around which is tied the filament of copper wire. In our preliminary report[14] it was stated that "microscopic examination of slides from the (first) experiment has revealed a complete absence of bone formation in the fistula." Additional slides studied since then

14. Guggenheim, P., and Guggenheim, L. K.: A New Method of Inhibiting Osteogenesis in Artificial Bone Fistulas: Preliminary Report, Arch. Otolaryng. **34**:356 (Aug.) 1941.

have revealed osteogenetic activity. Figure 1 *B* shows in the fistula a network of collagen and young connective tissue with cells having prominent nuclei. There are many distended vessels. Throughout the deposits of collagen are myriads of osteoblasts. Membrane bone is being actively formed everywhere. At the edges of the original bone are wide areas of new bone in process of formation. One gets the impression that within a short time the entire fistula will be filled solidly with bone.

Experiment 2.—Procedure: On Feb. 19, 1941, in a second rabbit a small fistula was made with a sharp chisel and treated with saturated copper sulfate solution. A large fistula was made nearby and left untreated. The experiment was terminated on August 2 (five months and eleven days later).

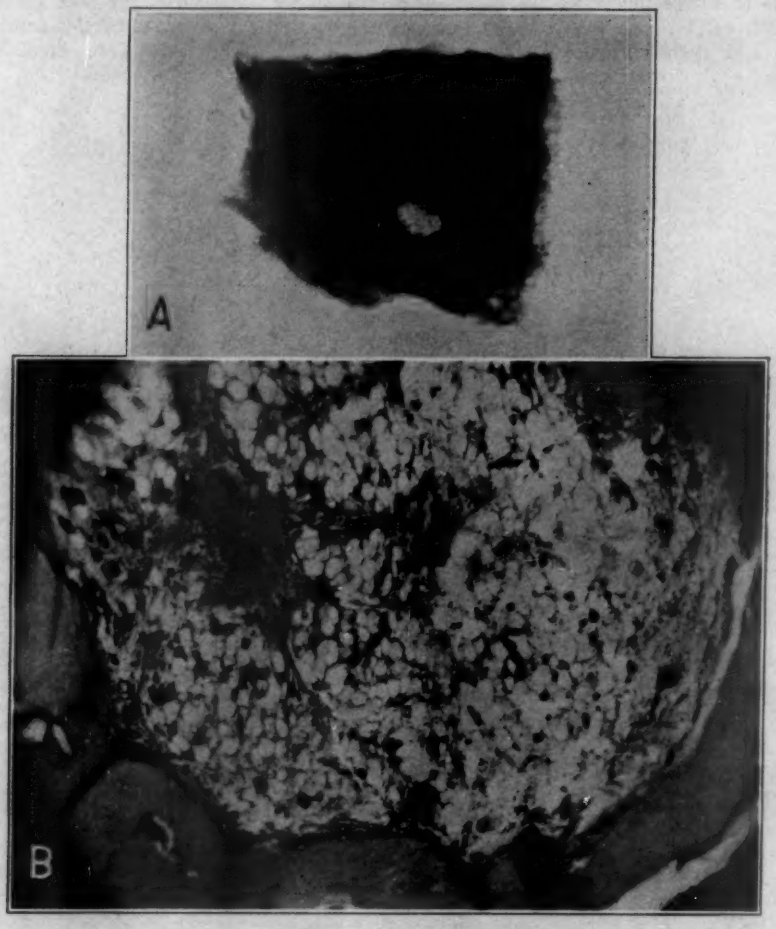

Fig. 3 (experiment 4).—*A*, roentgenogram of an osseous fistula treated with a 10 per cent silver nitrate solution. *B*, photomicrograph of the content of this fistula after four months and three days.

Result: The site of the first fistula was recognized grossly by dimpling of the bone. The fistula was apparently closed, resisting penetration by a sharp probe. The roentgenogram (fig. 2 *A*) shows the fistula to be closed with bone, but a suggestive oval area, possibly representing the fistula, is seen. The large control fistula closed without a trace and could not be identified.

Experiment 3.—Procedure: On Feb. 19, 1941 a large fistula with a centrally projecting ledge of bone was made with a sharp chisel, and a piece of copper wire was tied around the ledge. A small fistula made nearby was left untreated. The experiment was terminated December 5 (nine months and seventeen days later).

Result: The site of the first fistula was recognized grossly by a slight, darkened elevation of the overlying periosteum. The wire was not seen. The roentgenogram (fig. 2*B*) shows the wire in situ. The fistula is completely closed with bone. The control fistula could not be identified, having closed without a trace.

Experiment 4.—Procedure: On Aug. 2, 1941 the rabbit used in experiment 3 was subjected to the following additional procedure: Four additional fistulas were made with a burnishing burr, three in the vertex of the skull and one in the left nasal bone. One fistula

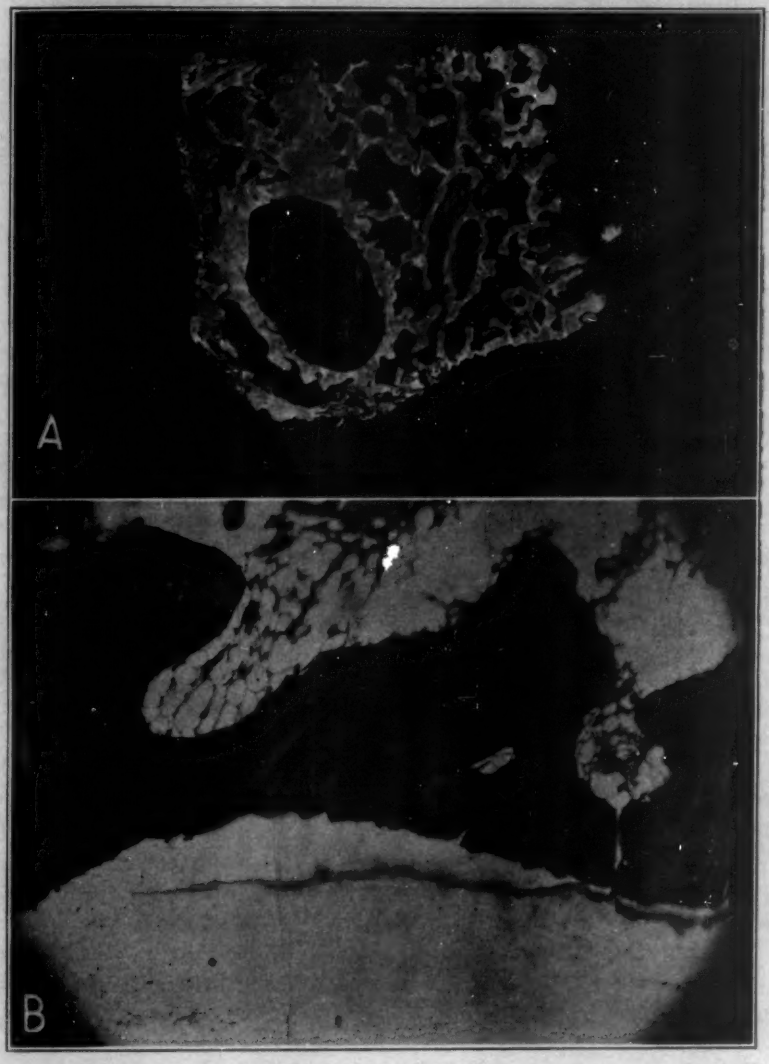

Fig. 4 (experiment 4).—*A*, osseous fistula treated with saturated solution of trichloroacetic acid. *B*, photomicrograph of the fistula after four months and three days.

in the vertex was treated with 10 per cent silver nitrate, one with saturated trichloroacetic acid solution and one with a 1:10 dilution of saturated copper sulfate solution. The fistula of the nasal bone was treated with a 1:100 dilution of saturated copper sulfate solution. Experiments 3 and 4 were terminated on December 5. The duration of the latter experiment was four months and three days.

Result in fistula treated with 10 per cent silver nitrate: The site was grossly blackened by a deposit of metallic silver. The fistula admitted a sharp probe. The roentgenogram (fig. 3*A*) shows a patent oval fistula. Microscopic examination of the fistula revealed

Data on Experiments with Chemical Agents for Maintaining Patency of Operative Fistula in Bone

Rabbit	Experiment	Character of Fistula	Technic: Fistula Made with	Location In	Agent	Duration of Experiment	Gross Results	Roentgen Results	Microscopic Results
1	1	Large, with centrally projecting ledge	Sharp chisel	Vertex of skull	Copper wire around ledge, saturated copper sulfate solution	2 mo., 11 days	Apparently patent	Apparently patent	Marked evidence of bony regeneration in fistula
2	2	Small	Sharp chisel	Vertex of skull	Saturated copper sulfate solution	5 mo., 11 days	Apparently closed	Closed	
		Large	Sharp chisel	Vertex of skull	None (control)	5 mo., 11 days	Not identified—apparently closed		
3	3	Large, with centrally projecting ledge	Sharp chisel	Vertex of skull	Copper wire around ledge	9 mo., 17 days	Closed	Closed, with copper wire in situ	
		Small	Sharp chisel	Vertex of skull	None (control)	9 mo., 17 days	Not identified (closed)		
	4	Medium	Burnishing burr	Vertex of skull	10 per cent silver nitrate solution	4 mo., 13 days	Patent	Patent	Dense collagen deposits (young connective tissue), impregnated with metallic silver; no new bone formation
		Large	Burnishing burr	Vertex of skull	Saturated trichloroacetic acid solution	4 mo., 13 days	Patent, filled only with loose fat	Patent	Fat dissolved in fixing, leaving empty fistula; no signs of bone activity
		Medium	Burnishing burr	Vertex of skull	1:10 dilution of saturated copper sulfate solution	4 mo., 13 days	Closed	Closed	
		Medium	Burnishing burr	Nasal bone	1:100 dilution of saturated copper sulfate solution	4 mo., 13 days	Not identified (closed)	Closed	

(fig. 3 *B*) a mesh of collagen deposits in which nuclei were dimly visible (very young connective tissue). Fat was abundant, the deposits being delimited by collagen membrane. Silver was deposited throughout, both in collagen and in fat. Nowhere in the collagen mass was there evidence of osteogenesis. At the osseous border the collagen was firmly attached. There were no deposits of silver in the bone. The bone surrounding the fistula was lamellar and dense; however, there were numerous marrow spaces (fatty). Nowhere did the marrow spaces or haversian canals communicate with the fistula. No osteoblasts were visible.

Result in fistula treated with saturated solution of trichloroacetic acid: The site was easily recognized. The bone edges were sharp, and the cavity was filled with loose fatty material. There was no evidence of caustic reaction at this time. Section showed a large, cleanly patent fistula (fig. 4 *A*). Microscopic examination revealed (fig. 4 *B*) an empty fistula surrounded by firm lamellar bone and large fatty marrow spaces. A few haversian canals and marrow spaces opened into the fistula. The bone edge was fairly smooth and devoid of any sign of osteogenesis. No osteoblasts or osteoclasts were visible.

Result in fistula treated with 1:10 solution of a saturated copper sulfate solution: The site of the fistula was identified only by a dimple in the bone. The roentgenogram (fig. 2 *C*) shows the fistula to be closed with bone.

Result in fistula treated with 1:100 dilution of a saturated solution of copper sulfate: The fistula was not identified, having closed without a trace.

COMMENT AND CONCLUSIONS

The experiments described do not indicate that there is a possibility of inhibiting osteogenesis in an artificial fistula of bone by means of selective chemical interference. A fistula can be kept patent by treatment with sufficiently caustic chemicals, and the degree of patency appears to vary with the degree of caustic action as follows: Copper sulfate, the least caustic, produces much fibrosis and delays bony regeneration. Silver nitrate, produces much fibrosis but successfully inhibits bony regeneration. Trichloroacetic acid, the most caustic, produces no fibrosis and inhibits completely bony regeneration.

It is highly doubtful whether the degree of injury to tissue necessary to produce a sufficiently patent fistula in this manner would be tolerated by the delicate labyrinth. Trichloroacetic acid, which gives the best technical result, is almost unquestionably too dangerous. Silver nitrate, because of the superficiality of its caustic action, might prove usable but for the fact that it produces an excessive amount of fibrosis within the fistula. Thus neither of these agents is practical. Between heroism and safety a satisfactory compromise does not appear feasible from this approach.

SUMMARY

Despite the fact that the fistula operation is today being performed with enthusiasm by a few otologists, it is pointed out that no one has yet succeeded in keeping patent an artificial fistula in an experimental animal with any method which could be employed safely in the wall of the labyrinth.

An attack on this problem was developed from biochemical considerations. An attempt was made to inhibit chemically the regeneration of bone within the fistula. The copper ion was believed to hold promise by virtue of its catalytic oxidizing effect on vitamin C, which is essential to the formation of the matrix of bone. Trichloroacetic acid and silver nitrate solutions were used as controls. The experiments are summarized in the accompanying table.

The results indicate that the end in view can be achieved, technically by the use of sufficiently powerful caustics but that specific interference with the biochemical mechanism of bone formation by copper is not demonstrated. The caustic agents which successfully prevent osteogenesis in experimental fistulas do not appear to be suitable for application to the labyrinth.

1033 Gayley Ave.

EFFECT OF SURGICAL TREATMENT OF THE SINUSES ON BRONCHIECTASIS

ANALYSIS OF RESULTS IN SEVENTY-FIVE CASES

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The purpose of this study is to present the results of operation on the paranasal sinuses which I have observed in 75 cases of bronchiectasis at the Massachusetts General Hospital and the Massachusetts Eye and Ear Infirmary, in order to determine the value of such treatment in the management of bronchiectasis. It is now so generally recognized that sinusitis is a common complication of bronchiectasis, if indeed it may not be a contributing etiologic factor at the onset in some cases, that rhinologists often look for notable improvement not only in the nose but, in general, in the infection in the chest. Unfortunately, the results are not often dramatic, or the hopes of the patient have been too high, so that not only the patient but the physician is disappointed, with a consequent tendency toward neglect of the sinuses as a part of the picture. I have chosen two criteria in estimating the postoperative improvement in the patient's health.

1. The local effect in the nose and sinuses. This is indicated by lessening of the discharge and return of the mucous membrane of the nose to a normal appearance.

2. The effect on the pulmonary symptoms. In general, this means a change in the amount of sputum and a change for the better in the patient's general vitality, even though complete relief from sputum is not obtained.

The results of operation on the sinuses are difficult to evaluate, inasmuch as the technic varies with different operators, the degree of infection is not the same in all cases and the individual response to operation differs. The last factor is especially important when the pulmonary infection is severe.

I have arranged the cases which form the basis of this analysis as follows:

Group 1. Cases with operation on the sinuses only.
Group 2. Cases with operation on the sinuses and lobectomy.
Group 3. Cases in which the sinuses were normal and lobectomy was performed.
Group 4. Cases in which lobectomy was performed and the sinuses were not operated on.

The results were estimated for the sinuses and for the chest separately. Four degrees of improvement in the postoperative condition are stated as follows:

1. Absence of symptoms. This means an absolutely normal nose and sinuses and no sputum.

2. Pronounced improvement. The patient may still have a slight nasal discharge or a small amount of sputum, but this condition represents a decided improvement over the preoperative situation. It also indicates a marked change for the better in the patient's capacity to work and to enjoy life.

3. Moderate improvement.

4. Slight or no improvement. The basis for assessment of the last two degrees of improvement is the same as that for pronounced improvement.

Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society, Inc., June 1942.

SUMMARY OF RESULTS

GROUP 1.—*Operation on sinuses only.*

In 15 cases there was bilateral pansinusitis, and in all but 1 case maxillary and ethmoid sinusitis was the minimum of infection of the sinuses. Radical operation on the antrum alone was done in 13 cases. In the other cases radical operations on the antrum and ethmoid cells were performed; in 2 cases radical external

TABLE 1.—*Results of Operation on the Sinuses Only in Thirty Cases of Bronchiectasis* *

	Result			
	Absence of Symptoms	Pronounced Improvement	Moderate Improvement	Slight or No Improvement
Sinuses	0	11	8	11
Chest	1	7	8	14

* In this table, and in the accompanying tables, results are expressed in terms of the number of cases.

TABLE 2.—*Results in Thirteen Cases of Bronchiectasis in Which Radical Operation on the Antrum Was Performed*

	Result			
	Absence of Symptoms	Pronounced Improvement	Moderate Improvement	Slight or No Improvement
Sinuses	0	1	5	7
Chest	0	3	5	5

TABLE 3.—*Results in Sixteen Cases of Bronchiectasis in Which Radical Operation on the Antrum and Ethmoidectomy Were Performed*

	Result			
	Absence of Symptoms	Pronounced Improvement	Moderate Improvement	Slight or No Improvement
Sinuses	0	10	2	4
Chest	0	5	5	6

TABLE 4.—*Results in Ten Cases of Bronchiectasis in Which Operation on Sinuses and Lobectomy Were Performed*

	Result			
	Absence of Symptoms	Pronounced Improvement	Moderate Improvement	Slight or No Improvement
Sinuses	3	5	2	0
Chest	0	7	2	1

operation on the frontal sinus and in 1 case bilateral operation on the antrum, ethmoid cells and sphenoid sinus were carried out. Of the cases in which improvement resulted, operation on the sinuses was not adequate in 5; in other words, there is still a sinus which should be treated surgically.

In table 2 are shown the results in cases in which radical operation on the antrum alone was done, ethmoiditis being left untreated. The results in these cases do not compare favorably with those in cases in which ethmoidectomy was also done (table 3).

As may be expected when no thoracic operation has been done, the greater improvement is local, with respect to the sinuses. Observations were made for a period of one to seven years after operation, an average of two and six-tenths years.

GROUP 2.—*Surgical treatment of the sinuses and lobectomy.*

In 2 cases in which there was complete relief from sinusitis, there was notable improvement in the chest also; in these cases observations were made for six and seven years respectively. In the case in which no improvement in the chest occurred the sinusitis was completely relieved.

As a control for the preceding cases, the results in 21 cases of lobectomy in which no sinus infection complicated the picture are shown in table 5. In other words, only one focus of infection needs consideration, that in the chest.

GROUP 3.—*Lobectomy and normal sinuses.*

It is evident that when lobectomy can be done in cases without sinusitis and the patient can overcome the immediate operative hazards, the results are very satisfactory.

TABLE 5.—*Results in Twenty-One Cases of Bronchiectasis in Which Lobectomy Was Done and the Sinuses Were Normal*

	Result			
	Absence of Symptoms	Pronounced Improvement	Moderate Improvement	Slight or No Improvement
Chest	7	14	0	0

TABLE 6.—*Results in Fourteen Cases of Bronchiectasis in Which Lobectomy But No Operative Treatment of the Sinuses Was Carried Out*

	Result			
	Absence of Symptoms	Pronounced Improvement	Moderate Improvement	Slight or No Improvement
Sinuses	0	3	0	11
Chest	1	10	1	2

GROUP 4.—*Lobectomy and no operative treatment of sinusitis.*

In the group of cases of bronchiectasis with sinusitis in which lobectomy was done and surgical treatment of the sinuses was not undertaken the results were less good (table 6). In these cases observations were made for one to eight years, the average period being three years. As might be expected, the results with regard to the sinuses were generally poor, but the fact that the chest failed to respond to surgical treatment as well as in the cases in which the sinuses were normal raises the question of persistence of sputum from unrelieved sinusitis. These cases should be compared with those in which both lobectomy and operation on the sinuses were performed (group 2).

COMMENT

Several points come up for discussion. First, the degree of infection in the sinuses and the chest is extremely variable. Some sinuses are slightly infected, and the degree of tissue change is too slight to warrant radical surgical intervention. There are all grades of pathologic change, from such slight involvement to severe pansinusitis, with reaction in the surrounding bone. Second, inasmuch as the general condition of the patient may contraindicate complete removal of patho-

logic tissue from all the sinuses which are affected, operation may fall short of the desired objective by just the extent to which it is incomplete. It would be unfair to state in such a case that the operation was a failure because the nose continues to discharge from an untreated sinus. The only failure lies in the fact that the patient could not stand a complete operation. Such a situation often explains the criticism of the patient and the general medical service that the operation did not bring the promised results. Third, it should be remembered that the conditions under discussion are chronic and of many years' standing and that the results must be viewed over a period of several postoperative years. It is not enough to judge by the results of the first few weeks. In my experience, there is frequently almost complete relief of sputum for the first days following an operation on the sinuses. It has been disquieting, therefore, to see the patient's record six months later and find that there is still sputum. However, a careful study over many months will show that the load of infection which the patient is bearing has been lightened. Even when there is persistent sputum, the removal of infection from the sinuses has improved the general health, so that there is a gain in weight and in strength and ability to carry on work.

SUMMARY

Seventy-five cases of bronchiectasis were studied to evaluate the importance of surgical treatment of the sinuses.

The best results were obtained from lobectomy in cases in which there was no complicating sinusitis.

Untreated sinusitis was a definite hindrance to recovery from the thoracic infection.

Likewise, in cases in which the sinuses were operated on but lobectomy was not done the result was poor.

When both adequate operation on the sinuses and lobectomy can be performed the results are so much better that these two procedures are indicated in all suitable cases.

That the cases were hard to classify may be considered evidence that each patient must be considered individually.

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ESTIMATION OF PERCENTAGE OF COMPENSABLE HEARING DEFECTS

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The percentage estimation of a hearing defect due to accident or occupation is a question which concerns industry, insurance companies, courts of law and compensation boards. With the United States at war, this question will also be of vital concern to the Government when the armed forces are demobilized. After about four years of work and after studying and discarding various methods proposed, the Committee of Consultants on Audiometers and Hearing Aids of the Council on Physical Therapy of the American Medical Association proposed a tentative method of estimating these hearing defects in percentages which was approved by the Section of Otolaryngology and the House of Delegates, adopted by the Council on Physical Therapy and published in *The Journal of the American Medical Association*.[1]

With the realization that no means of estimating these hearing defects in percentages can be perfect, and in the hope that future study may show some way of improving the one proposed, the method was put forward as a tentative procedure. It is palpably impossible to construct a single table or method which will operate equitably for all concerned. Some persons need much better hearing for their occupations than others. The musician, the piano tuner, the stenographer, the telephone operator, the navigator in fog-bound seas and the officer in charge of the sound detector of a submarine need much sharper hearing than the farmer or the laborer in the heavy industries. The hearing requirements of these specialized workers are variables which cannot be included in a generalized table, but must be specially considered by judicial bodies. It was, therefore, decided by the Committee of Consultants to devise a table or method more or less applicable to all, without consideration of cultural requirements or the requirements of specialized occupations, these special problems being left to the discretion of courts and juries. A clear distinction must be drawn between the loss of hearing expressed in percentages and the disability resulting therefrom. The method proposed is not a disability table in any sense of the word.

For the construction of a percentage table applicable to cases of traumatic loss of hearing, the ability to hear and understand the human voice was taken as a criterion.

Inasmuch as the use of an audiometer for the measurement of hearing defects has now become rather universal among otologists, and inasmuch as such instruments can be kept properly calibrated, the accepted standard audiometer has been designated as the instrument with which the hearing defect shall be measured.

Crowe [2] took exception to the fact that the committee saw fit to use the expression "an accepted standard audiometer" instead of "a correctly calibrated and

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Read at the meeting of the Middle Section of the American Laryngological, Rhinological and Otological Society, Inc., Detroit, Jan. 20, 1943.

1. Tentative Standard Procedure for Evaluating the Percentage of Useful Hearing Loss in Medicolegal Cases, report of the Council on Physical Therapy, J. A. M. A. **119**:1108 (Aug. 1) 1942.

2. Crowe, S. J., Editorial Remarks, in Bothman, L., and Crowe, S. J.: The 1942 Year Book of Eye, Nose and Throat, Chicago, The Year Book Publishers, Inc., 1942, p. 395.

properly used audiometer." He made the comment that "the fact that a certain audiometer has been approved by the American Medical Association or any other organization does not mean that all hearing tests made with the instrument of this make and model are reliable." This is perfectly true, but it is known that when an audiometer has been accepted by the Council on Physical Therapy it has met the minimum requirements of the Council, and it is assumed that the user will keep it properly calibrated and make the hearing tests in the proper manner.

Inasmuch as the loss of ability to hear the human voice was taken as a criterion of traumatic hearing defects by the committee, it was necessary to define "usable hearing" and to determine what constitutes a total, or 100 per cent, loss of such usable hearing. This was arrived at in the following way:

Dr. C. C. Bunch, a member of the committee, exhibited (1) a number of audiograms of persons who could just hear and understand words in a familiar language shouted close to the ear and (2) audiograms of persons who could hear the words so shouted but could not understand them. He also presented scattergrams of the audiograms of each group and constructed a chart showing two lines on the audiogram, namely, *A*, those who could understand the words and, *B*, those who could not. A line, *C*, was drawn equidistant from the lines *A* and *B*, which represented a compromise between the two. This line *C* was taken as indicating a total, or 100, per cent loss of "usable hearing." This line is indicated by the following losses at the individual frequencies:

128	256	512	1024	2048	4096	cycle/sound
65	80	85	95	95	90	decibels

This line does not represent the total loss of all hearing but does indicate the total, or 100 per cent, loss of the ability to hear and understand speech. This empiric line, *C*, adopted by the committee, did not show more than a 5 to 10 decibel difference at each frequency as compared with a similar line established by Dr. Steinberg, of the Bell Telephone Laboratories.

In establishing a threshold for usable hearing, it was felt that the zero line on audiograms was too high and that most persons with an audiometric loss of 10 decibels still had normal hearing for speech. The 10 decibel line was, therefore, taken as the threshold (fig. 1).

In agreement with the previous work of such investigators as Fletcher, Steinberg, Fowler, Bunch and Sabine, it was felt that all tones or frequencies between 256 and 4096 cycles were of value for the understanding of speech. Also, in agreement with these men, it was believed that the frequencies or bands of frequencies between 1000 and 3500 cycles were relatively more important than the others. It was also felt that frequencies below 256 and above 4096 cycles had no particular value for the understanding of speech.

Therefore, in apportioning of the audiometric field between 256 and 4096 cycles, more relative importance or weighting was assigned to these bands of frequencies than to that portion of the field below 1000 and above 2048 cycles. However, as the interest lay in the ability to hear the entire band of frequencies between the octave lines, and not merely the pure tones of each octave, the weighted values chosen were assigned not to the octave lines themselves but to the spaces or rectangles between these lines. As can be seen on the audiogram, the relative value for each of the four spaces increases from the value assigned to the space between 256 and 512 cycles to the value assigned to the space between 1024 and 2048 cycles and then decreases.

It was also felt that the relative value of a hearing defect increases from threshold to about a 50 decibel loss and then decreases. Therefore, the increment in value for each rectangle increases up to the 50 decibel line and then decreases.

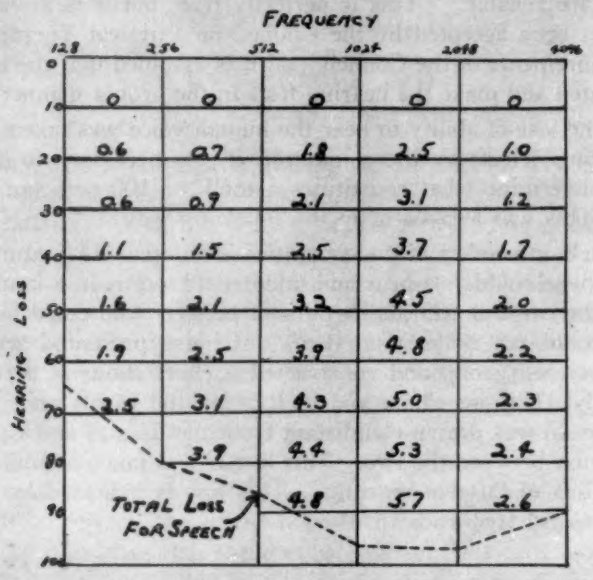

Fig. 1.—Original weighting of the audiometric field (from Sabine, P. E.: On Estimating the Percentage Loss of Useful Hearing, Tr. Am. Acad. Ophth. [1941] **46**:179 [March-April] 1942).

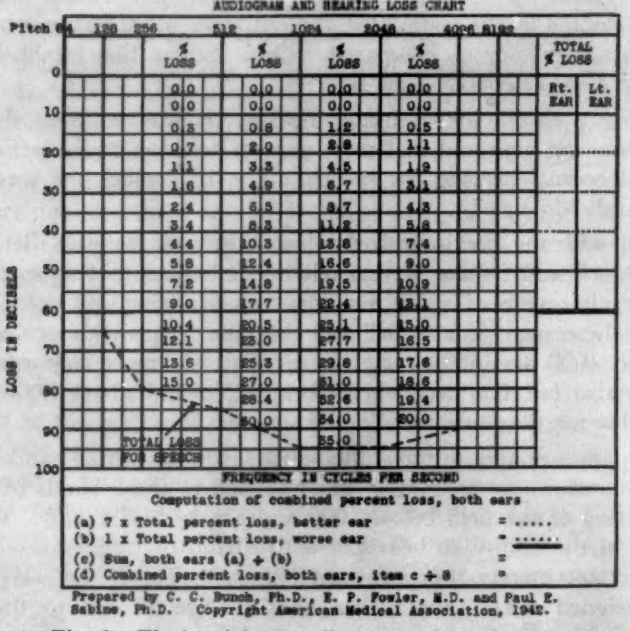
AUDIOGRAM AND HEARING LOSS CHART
Pitch 64 128 256 512 1024 2048 4096 8192
% LOSS
% LOSS
% LOSS
% LOSS
TOTAL % LOSS
Rt. EAR
Lt. EAR
LOSS IN DECIBELS
0
10
20
30
40
50
60
70
80
90
100
0 0 0 0 0 0 0 0
0 0 0 0 0 0 0 0
0 3 0 8 1 2 0 5
0 7 2 0 2 8 1 1
1 1 3 3 4 5 1 9
1 6 4 9 6 7 3 1
2 4 6 5 8 7 4 3
3 4 8 3 11 2 5 8
4 4 10 3 13 8 7 4
5 8 12 4 16 6 9 0
7 2 14 8 19 5 10 9
9 0 17 7 22 4 12 1
10 4 20 5 25 1 15 0
12 1 23 0 27 7 16 5
13 6 25 3 29 8 17 6
15 0 27 0 31 0 18 6
28 4 32 6 19 4
30 0 34 0 20 0
35 0
TOTAL LOSS FOR SPEECH
FREQUENCY IN CYCLES PER SECOND
Computation of combined percent loss, both ears
(a) 7 x Total percent loss, better ear = ...
(b) 1 x Total percent loss, worse ear = ...
(c) Sum, both ears (a) + (b) =
(d) Combined percent loss, both ears, item c ÷ 8 =
Prepared by C. C. Bunch, Ph.D., E. P. Fowler, M.D. and Paul E. Sabine, Ph.D. Copyright American Medical Association, 1942.

Fig. 2.—Final weighted audiogram and hearing loss chart.

For simplicity in computation of the percentage loss, the figures seen in each rectangle represent not the value for that particular rectangle alone but, rather, the combined values for that rectangle and all those above it.

In computation of the percentage loss for each ear, the successive points on an audiogram are connected by straight-ruled lines. "The percentage loss to be assigned to each octave interval is the figure in the space immediately above the straight line drawn in that octave." The report states that "when two or more spaces are intersected, the average of the figures in the intersected spaces is to be taken." This, to my mind, is not quite correct, because, in arriving at the percentage loss for the band of frequencies in that particular column, the figure in the space just above the highest point on either side of the column should be included with the figures of the intersected spaces in determining the average percentage loss of that particular column. For example, if one draws a line from 40 decibels at 512 cycles to 45 decibels at 1024 cycles, one half of the intersected space would be 5.2 per cent, whereas there already is an 8.3 per cent loss at 40 decibels. One should, then, average 8.3 and 10.3 per cent, which gives a value of 9.3 per cent for that column.

While the computation of a monaural loss of hearing by this method is based on the accumulated data of such investigators as Fletcher, Steinberg, Fowler, Knutsen, Bunch and Sabine, the computation of binaural deafness has been arrived at on a more or less empiric basis. The weighting of the percentage loss for the better ear as compared with that for the poorer ear on the basis of 7 to 1 represents the composite judgment of the committee based on clinical experience and practical considerations. This method of computation means that a subject with a normal ear and a completely deaf ear will have lost 12 per cent of his total hearing capacity. The committee would, I know, welcome any concrete suggestions on this phase of its report.

This procedure for determining the percentage loss of ability to hear and understand speech represents the more or less continuous work of a group of otologists for three or four years. It is not presented as a final solution of the problem but is intended, rather, as a "tentative standard procedure." While the method is a far cry from perfection, it does represent a distinct improvement on the confusion which has hitherto existed. Heretofore, when a group of otologists appeared before a judicial body, no two of them were ever able to agree on how much loss of hearing there was, expressed in percentages.

The method estimates, in a general way, the percentage loss of hearing a person has suffered but does not attempt to evaluate the disability accruing therefrom. Such an estimate of disability is a function of courts, juries and other judicial bodies. When this method is made to apply to persons in specialized occupations, it is the function of these judicial bodies to determine in how far it should apply. This matter of compensable disability is certainly no direct concern of the otologist.

DICED CARTILAGE GRAFTS

NEW METHOD FOR REPAIR OF SKULL DEFECTS, MASTOID FISTULA AND OTHER DEFORMITIES

LYNDON A. PEER, M.D.
NEWARK, N. J.

In 1938 I reported on the operative repair of deep depressions in the frontal region of the skull in 15 patients. The depressions were filled with segments of autogenous rib cartilage, inserted one above the other, as illustrated in figure 1. These patients presented normally rounded skull contours until about two months after the operation, but from this time on irregularities developed which outlined the separate rib cartilage segments. The irregularities were most noticeable when an extensive depression had been repaired.

Secondary operations in which projecting portions of cartilage were removed served to improve the line of the forehead, but did not completely correct the irregularities. Some immediate improvement was also obtained by introduction of sheets of dermal graft between the cartilage and the overlying skin of the scalp, but the late results were disappointing. Depressions in the skull repaired by insertion of bone grafts likewise present postoperative irregularities, similar to those seen after the introduction of segments of rib cartilage.

DICED CARTILAGE GRAFTS IN REPAIR OF DEPRESSIONS IN THE SKULL

About five years ago it occurred to me that cartilage in small segments might lend itself to smoother molding. The cartilage could be cut into many fine squares, much as a cook dices carrots when preparing a salad. These fine cartilage squares could then be introduced into an exposed depression in the skull and gently patted into a rounded contour and the skin of the scalp sutured over the rounded surface of the cartilage mass. I repaired a rather deep depression in the skull in this manner and was extremely pleased with the simplicity of the procedure and with the postoperative result (fig. 3).

Since then I have repaired depressions in the frontal region of the skull by this diced cartilage method in 7 cases. The postoperative contours of the forehead in these cases have been far superior to those previously obtained with large segments of rib cartilage.

I experimented to learn what change or changes in the diced cartilage took place in situ. I stored the excess of diced rib cartilage beneath the skin of the chest of 7 patients. After successful operation on the skull the stored excess cartilage was removed at intervals and sectioned. Microscopic examination showed that the spaces between the small cartilage squares were occupied first by blood and later by ingrowing connective tissue, accompanied by numerous blood vessels. Each small square of cartilage rested against adjoining cartilage squares at some points, contracture of the cartilage mass being thus prevented. The filling in of

Presidential address delivered at the annual meeting of the American Society of Plastic and Reconstructive Surgery, Baltimore, Dec. 4, 1942.

the numerous small spaces between the individual cartilage grafts with connective tissue added to the bulk of the entire diced segments. For this reason the diced cartilage grafts actually filled a larger space than did the solid rib cartilages from which they were cut.[1]

Autogenous diced rib cartilage grafts showed well nourished cells, normal-appearing matrix and complete absence of invasion or absorption (fig. 8). Preserved diced rib cartilage grafts showed definite invasion and partial absorption of the cartilage.

Diced cartilage grafts should be formed from the patient's own rib cartilage whenever possible.[1a] Rib cartilage from a near relative is second choice, and diced cadaver rib cartilage preserved in merthiolate [2] or alcohol is third choice. Autogenous cartilage grafts are indicated for children, who, because of their long life expectancy, require longer-lasting grafts than do older persons. Autogenous

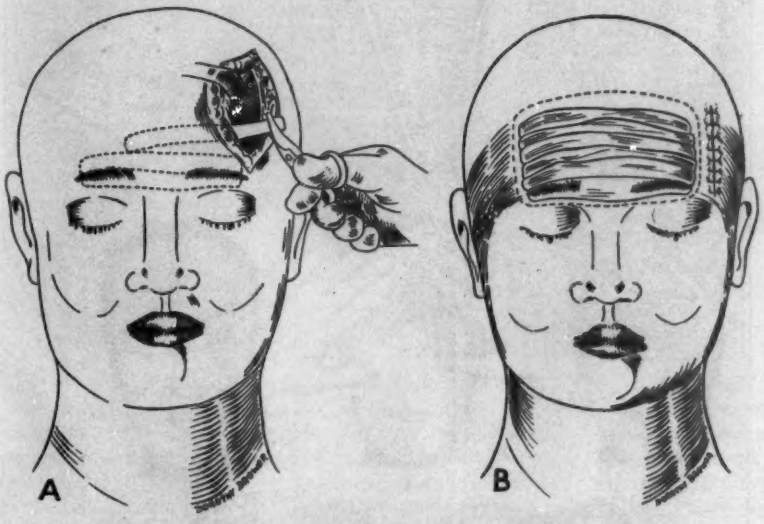

Fig. 1.—Diagrams illustrating the usual method of inserting large segments of rib cartilage to fill a depression in the frontal region of the skull. The diagram at the left shows insertion of rib cartilage; the diagram at the right, the position of the rib cartilage, frontal view. After operation uneven contracture produces irregularities in the contour of the forehead. These irregularities do not occur when diced cartilage grafts are used instead of large segments of cartilage (see fig. 2). From Peer (Types of Buried Grafts Used to Repair Deep Depressions in the Skull, J. A. M. A. **115**:357-360 [Aug. 3] 1940, figs. 5 and 6).

cartilage grafts are also desirable for children because of the growth possibilities of young living cartilage grafts.[3] Preserved dead cartilage or living cartilage

1. In experimental work with dogs, Young noted that separate cartilage segments became joined together by fibrous tissue after transplantation (Young, F.: Surgery **10**:7, 1941).

1a. Peer, L. A.: The Fate of Living and Dead Cartilage Transplanted in Humans, Surg., Gynec. & Obst. **68**:603-610 (March) 1939.

2. Pierce, G. W., and O'Connor, G. B.: Reconstruction Surgery of the Nose, Ann. Otol., Rhin. & Laryng. **47**:437-452 (June) 1938.

3. Dupertuis, S. M.: Actual Growth of Young Cartilage Transplants in Rabbits, Arch. Surg. **43**:32-63 (July) 1941.

from a relative may be used in older persons or in any patient whose general condition precludes the removal of his or her own rib cartilage.

OTHER CLINICAL USES OF DICED CARTILAGE GRAFTS

I have used diced autogenous rib cartilage grafts to form the structural support in 5 complete reconstructions of the ear. This method permits the surgeon to form a cartilaginous plaque of any desired size for the contour and support of the new ear.

Several rib cartilage segments are removed from the right side of the child's chest and diced into many small squares. A pocket of the desired size is then

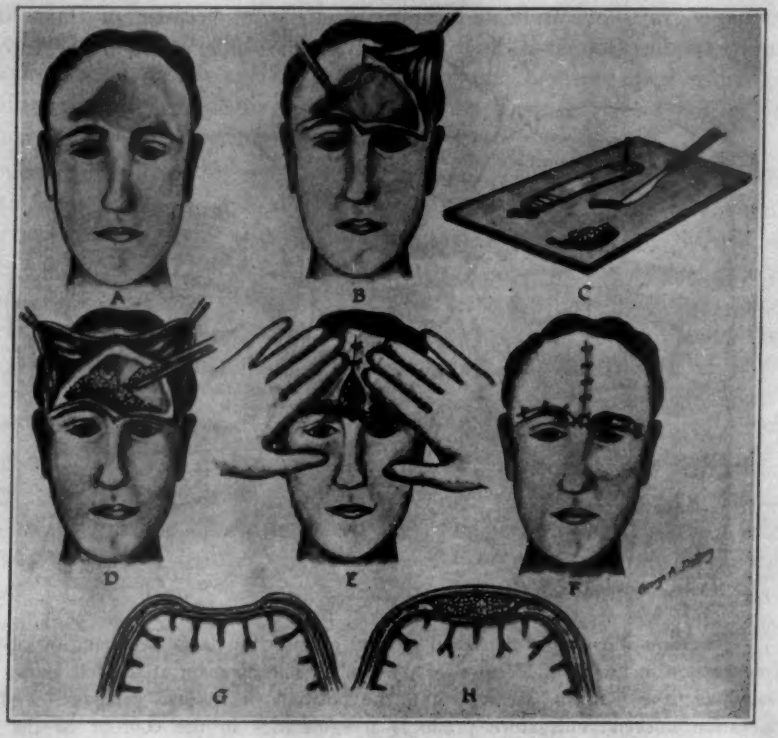

Fig. 2.—Repair of a depression in the skull with diced cartilage grafts. *A*, deep depression in the frontal region of skull. *B*, exposure of a skull defect by an inverted T incision. *C*, diagram illustrating the dicing of rib cartilage, which has been removed from the right side of the patient's chest. This may be done by an assistant while the surgeon is exposing the depression in the skull. Fresh rib cartilage from a relative or preserved rib cartilage from a cadaver may be used instead of autogenous rib cartilage. *D*, introduction of diced cartilage grafts into a depression in the skull. *E*, method of gently patting the mass of diced cartilage squares into a rounded contour after the upper part of the midline incision has been sutured. Excess diced cartilage grafts are squeezed out through the lower portion of the incision. *F*, skin of scalp completely sutured over the rounded surface of the diced cartilage grafts. On the left side the incision should extend through, instead of below, the brow. *G*, diagrammatic cross section in the region of the skull defect before introduction of diced cartilage grafts. *H*, diagrammatic cross section in the region of the skull defect after introduction of diced cartilage.

formed beneath the skin of the scalp and the diced cartilage packed in this pocket. During the course of a few weeks connective tissue and blood vessels invade the

Fig. 3.—*A*, photograph of a patient with a deep bony defect extending through the left brow and the frontal bone above the brow. The upper eyelid is completely everted and drawn upward into the depression. The defect extended down to the dura, but the left frontal sinus was not involved. *B*, another preoperative photograph, showing a second, smaller depression on the right side of the forehead. *C*, lateral view three years after the defect had been filled with diced autogenous rib cartilage. The everted eyelid was repaired with a split skin graft taken from the thigh. *D*, frontal view three years after operation. The smaller depression above the right brow was also filled with autogenous diced cartilage grafts. Note the even contour of the forehead and the left brow.

Fig. 4.—*A*, deep depression in the right brow, the root of the nose and the adjacent frontal bone, extending down to the dura. Dotted lines indicate the extent of the depression. *B*, photograph taken two and a half years after operative repair. The depression was filled with diced autogenous rib cartilage grafts.

Fig. 5.—*A*, depression following operation to expose and drain an abscess in the frontal lobe. The bony portion of the left brow, the left frontal sinus and a moderate amount of the adjacent frontal bone had been removed. *B*, photograph taken three years after the depression had been filled with diced autogenous rib cartilage grafts, and after a secondary operation to remove scar tissue from the left brow.

Fig. 6.—*1*, photograph of patient, with a deep depression in the right frontal region following removal of the bony portion of the brow, the frontal sinus and a large adjacent area of frontal bone. *A* indicates thin scar tissue covering the frontal lobe of the brain; *B*, the retracted skin of the forehead. *2*, photograph taken three days after operation to illustrate the method of providing a complete covering of skin over the cartilage inserted to fill the defect. The thin layer of epidermis at *A* (in *1*) was carefully dissected from the scar tissue covering the brain. The retracted skin of the forehead was then undermined and sutured as far upward as possible and a skin flap brought downward from the scalp to cover the remaining open area. *B* represents the skin of the forehead, and B^1 the scalp flap. A later postoperative photograph was not obtained.

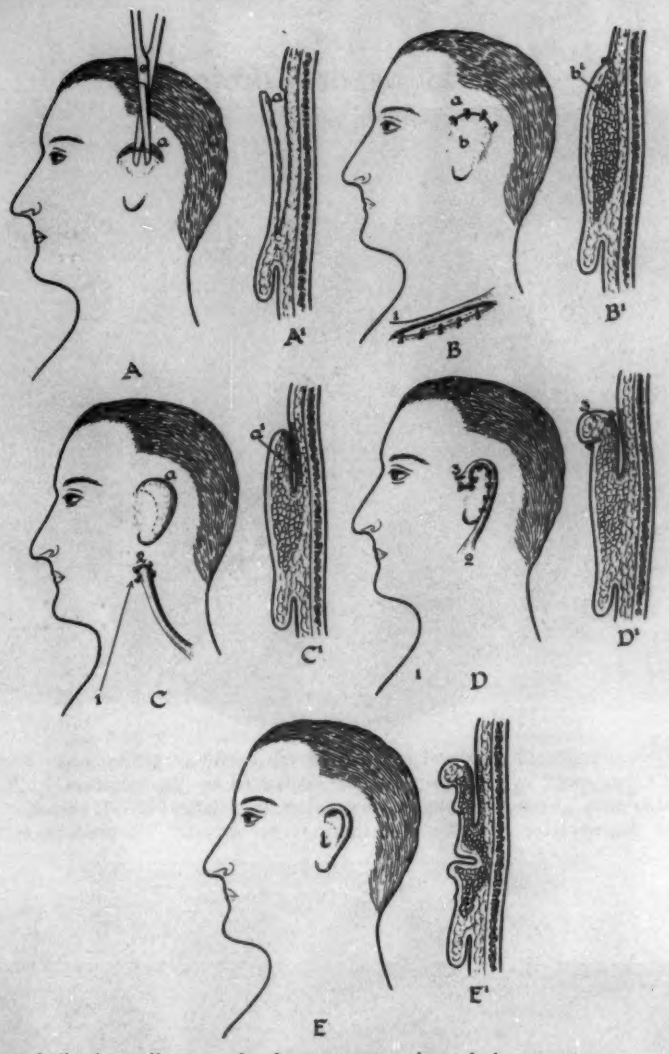

Fig. 7.—Use of diced cartilage grafts for reconstruction of the ear.

(*A*) A pocket is formed beneath the skin of the scalp through an incision somewhat higher than the top of the normal ear on the other side. *A* 1 represents a cross section, showing the subcutaneous pocket at *a*.

(*B*) Several segments of rib cartilage have been removed from the right side of the patient's chest and diced into many fine squares. The mass of diced cartilage is then packed into the subcutaneous pocket, and the wound is sutured. At the same operation a small tube flap is formed over the patient's clavicle. *B* 1 represents a cross section, with the mass of diced cartilage grafts indicated by *b* 1.

(*C*) About three weeks later the plaque of diced cartilage grafts is separated on its inner surface from the underlying tissue, and a dental stent impression is made of the cavity. A split skin graft is then wrapped about the dental stent, and the stent, with its covering of skin, is introduced into the cavity. Dotted lines at *a* indicate the dental stent, with its covering of skin in position behind the cartilage plaque. During the operation one end of the tube flap of skin is detached from its original position, at *1*, and attached higher up, at *2*. It is safer to delay the tube before each transfer. *C* 1 represents a cross section, showing the split skin graft lining the deep surface of the cartilage plaque and the scalp surface of the pocket. This permits the ear to stand out from the head.

(*D*) The lower end of the tube flap has been severed and is shown attached at *3* to the upper and posterior margins of the cartilage plaque to form the helix of the new ear. *D* 1 represents a cross section, showing the tube flap attached to the cartilage plaque at *3*.

(*E*) The excess portion of the tube flap has been discarded and the diced cartilage plaque carved out to simulate the external auditory canal, the tragus and the other adjacent structures of the ear. Unfortunately, one cannot make as perfect an ear as the artist has illustrated. *E* 1 represents a cross section showing the portions of the cartilage plaque removed to simulate the normal structures of the ear. A full thickness skin graft removed from behind the normal ear may be used to replace hair-bearing skin on the external surface of the reconstructed ear.

Fig. 8.—Photomicrograph of diced autogenous rib cartilage grafts which have been buried three years. Three diced cartilage squares are indicated by the numbers *1, 2* and *3*. *CT* is the intercartilaginous connective tissue, containing two large blood vessels. The cartilage cells show no degenerative changes, and there is no invasion or absorption of the hyaline matrix.

Fig. 9.—*A*, a child, aged 4 years, who was born with complete absence of the right auricle except for a misplaced ear lobe. This photograph shows a late stage of the reconstruction, with a skin-lined pocket behind the framework of autogenous diced cartilage and a tube of skin which has been brought up from the region of the clavicle.

B, completed auricle, with the tube of skin used to form the helix and the diced cartilage framework carved to simulate the external auditory canal and the adjacent structures of the ear. Diced cartilage may be packed to form a thick cartilage mass in the region of the external auditory canal, which provides sufficient bulk for later carving.

Fig. 10.—*A*, boy aged 10 years who was born with complete absence of the right auricle. Note the distorted cartilage in the region of the ear. This patient also had pronounced atrophy of the right side of his face. *B*, reconstructed right auricle, using autogenous diced rib cartilage for the framework. Note the atrophy of the right side of the face.

Fig. 11.—Boy aged 11 years who was born with complete absence of the right auricle except for a misplaced ear lobe. The photograph shows a late stage in the reconstruction, with a skin-lined pocket behind the autogenous diced cartilage framework and a tube of skin which has been elevated from the region of the clavicle. The upper two thirds of the skin of the scalp covering the graft was hair bearing. This hair-bearing skin has been excised and replaced by a full thickness skin graft removed from behind the opposite ear.

In this case the reconstruction has not been completed.

intercartilaginous spaces, binding the small cubes together in the form of a solid, elastic plaque. Another method is to form the cartilaginous plaque beneath the child's abdominal skin and at a later operation transplant the whole plaque beneath the skin of the scalp.

The remaining steps, in general, follow the Pierce technic [4] for total reconstruction of an external ear. I have found it advantageous to pack the diced cartilage rather thickly in the lower part of the scalp pocket. This provides a bulky portion in the cartilaginous plaque, which can later be carved out to simulate the external auditory canal, the concha and the adjacent structures of the ear.

Fig. 12.—Drawings illustrating how a depression over the malar bone may be repaired with diced cartilage grafts. Depressions of the bony floor of the orbit and depressions in the mastoid may also be filled with diced cartilage grafts.

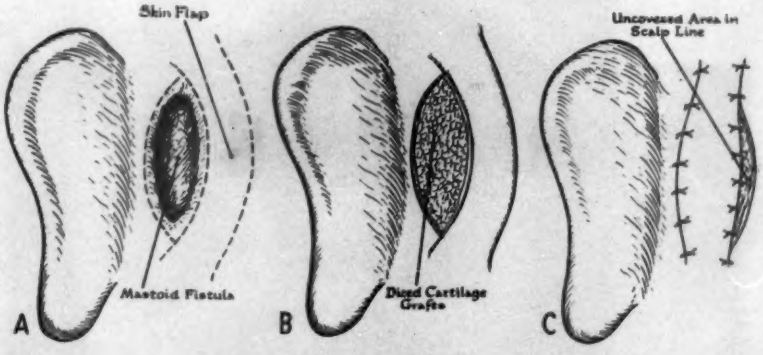

Fig. 13.—Repair of mastoid fistula with diced cartilage grafts.

(*A*) An elliptic incision is made about the fistula. The thin scar tissue within this section is then removed, with exposure of the bony surface of the depression in the mastoid. A skin flap formed behind the fistula is indicated by dotted outline.

(*B*) The mastoid fistula has been filled with diced cartilage grafts to obliterate the depression and provide a base for the skin flap. Preserved cadaver cartilage is an adequate filling substance for a mastoid fistula.

(*C*) The skin flap is brought forward and sutured to cover the depressed area filled with diced cartilage grafts. The small, uncovered area behind the flap heals by granulation and is not noticeable because it is located within the hair line. The skin flap may be outlined two weeks before it is brought forward to cover the defect in order to insure an adequate blood supply.

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4. Pierce, G. W.: Reconstruction of External Ear, Surg., Gynec. & Obst. **50**:601-605 (March) 1930.

Hair-bearing skin on the outer surface of the new ear is excised and replaced by a full thickness graft of auricular skin taken from behind the child's other ear. Because of the rich blood supply in the intercartilaginous spaces, such a graft will take well even though placed directly in contact with the cartilaginous plaque.

Diced cartilage grafts may also be used to fill depressions over the malar bone or to elevate a depressed floor of the bony orbit. They form an ideal filling substance for a deep depression in the mastoid resulting from an extensive simple mastoidectomy.

965 Broad Street.

Case Reports

MIXED TUMOR OF THE SOFT PALATE

SIDNEY H. GIDOLL, M.D., SAN FRANCISCO

According to Fetissov,[1] opinions differ regarding the histogenesis and histopathology of endotheliomas, but such growths may be considered to have a definite histologic structure and should be strictly differentiated, particularly from the so-called mixed tumors grouped with them. On the other hand, Volkman, cited by MacCallum,[2] on the basis of a study of many cases of mixed tumor, decided that the narrow strands of cells were derived from endothelium, and such tumors of the parotid gland made up a great part of the material for his monograph on endotheliomas.

It is therefore not surprising that in the case now under discussion the microscopic diagnosis of angioendothelioma had been made by one pathologist and that of mixed tumor of the salivary gland by two others. Because of the comparative rarity of mixed tumors in the soft palate (Sonnenschein [3]) and the confusion and contradictions in the nomenclature of pharyngeal growths (Salinger and Pearlman [4]), a report of this case is thought pertinent.

Endothelioma may develop wherever there are blood and lymph vessels, especially in the skin, the subcutaneous tissue, the cerebral meninges, the tissues of the oral cavity and the tissues of the hard and soft palate. A mixed tumor of the salivary glands is a rounded or nodular elastic mass, which grows, as a rule, not in the gland but in close proximity to it, being generally attached to its capsule. The tumor springs in this way from the parotid or the submaxillary gland and may reach great size. On extirpation it may recur, but even then it runs a benign course. On section, such a tumor presents extensive, rather translucent, areas, with occasional patches of denser opaque tissue and rare points of calcification.

The histologic structure of endothelioma is characterized by polymorphism. In the same specimen there may be flat, cubical and longish cells, as well as other forms. The relation between the connective tissue stroma and the parenchyma differs greatly; in some tumors the one type of tissue predominates, and in other tumors, the other type. Another characteristic is the tendency toward degeneration of various types, particularly the hyaline. On the other hand, the microscopic appearance of mixed tumors is most varied. The stroma is hyaline, or like the matrix of cartilage; there may be true cartilage, mingled with dense fibrous tissue. Everywhere there are masses of cells arranged in tubules or cysts, or in long tapering strands which anastomose and finally fade into the crevices of the stroma. In some cases, but not in all, patches of this cellular tissue are distinctly and unmistakably composed of stratified epithelium.

REPORT OF CASE

A white man aged 44 was seen April 10, 1936, with the complaint of a "gum boil" in his mouth. He had been aware of its presence for the past three weeks, but was not certain how long prior to that it had existed. Only slight soreness and very slight interference with swallowing were noted; there was no hemoptysis, no dyspnea, no nasal obstruction and no

1. Fetissov, A. G.: Endothelioma of the Pharynx, Arch. Otolaryng. **23**:712 (June) 1936.
2. Volkman, cited by MacCallum, W. G.: Text-Book of Pathology, Philadelphia, W. B. Saunders Company, 1940.
3. Sonnenschein, R.: Mixed Tumors Occurring in the Soft Palate, with Reports of Two Cases and a Survey of the Literature, Arch. Otolaryng. **10**:561 (Nov.) 1929.
4. Salinger, S., and Pearlman, S. J.: Malignant Tumors of the Epipharynx, Arch. Otolaryng. **23**:149 (Feb.) 1936.

earache. His physician attempted to lance the swelling, surmising it to be an encapsulated abscess, but no relief followed this procedure. No unusual bleeding ensued, although the soreness became slightly more pronounced.

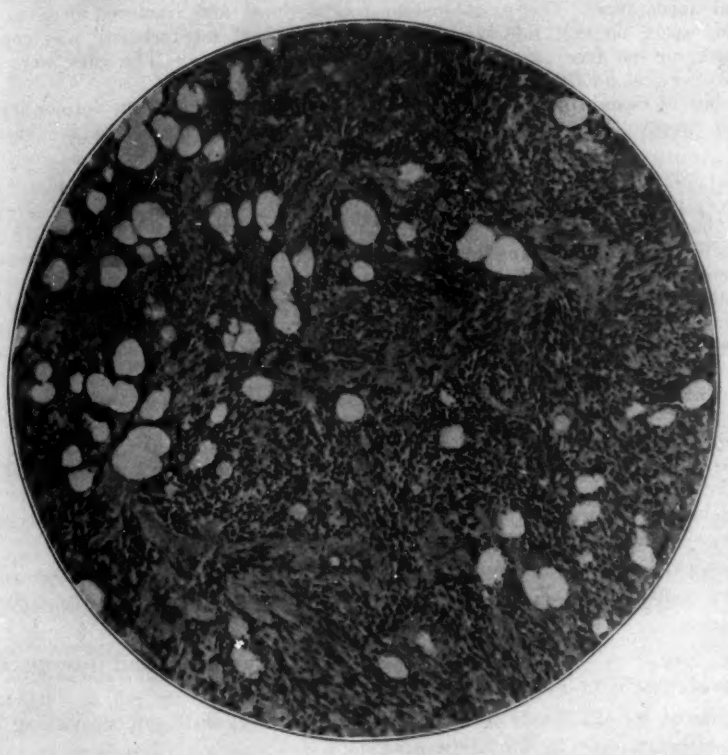

Fig. 1.—Photomicrograph of mixed tumor of the salivary gland, showing mucinous degeneration of the stroma with an epithelial sweep of cells and polyglandular arrangement.

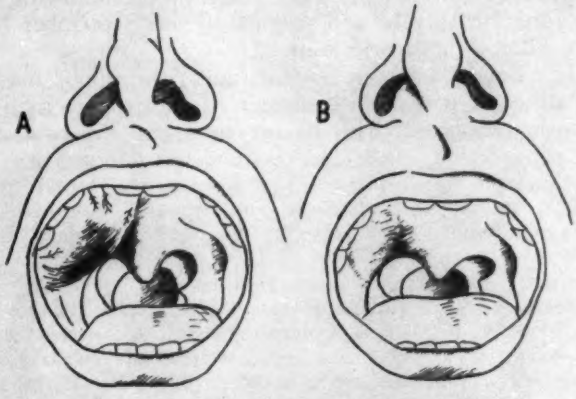

Fig. 2.—*A,* benign mixed tumor of the salivary gland, occupying the right half of the soft palate. *B,* appearance and size of a mixed tumor of the soft palate after radium therapy.

Examination.—A large, round, somewhat fluctuant, mass, 3.5 by 5 cm., occupied the right half of the soft palate, directly above and anterior to the right palatoglossus muscle. Several small vessels radiated toward the center from the periphery, and a small, indented depression in this mass, near the right supratonsillar area, indicated the site of the previous exploratory

puncture. There was no tenderness on superficial or deep pressure, and the consistency of the swelling could be more accurately described as "rubbery."

The tonsils were buried and fibrous and there was good dental hygiene. Indirect laryngoscopic examination revealed that the vocal cords and the arytenoid cartilages were of normal motility and appearance. The nasal septum was deviated and fractured over to the right, the breathing space on that side being poor. The left inferior turbinate was engorged and hypertrophied, but no free pus was in evidence on either side. The ears were essentially normal. No cervical adenopathy was present.

Several bits of tissue from the tumor mass adjacent to the site of the former incision were removed for biopsy. There was surprisingly little bleeding, considering the vascularity of the mass.

Laboratory Data.—The patient's temperature was 37 C. (98.6 F.), his pulse rate 78 and his respiratory rate 16. The Kolmer and Kahn reactions of the blood were negative; examination of the blood and urine revealed essentially nothing abnormal. On April 15 the biopsy report from the pathologic laboratory stated: "Microscopic sections showed a densely vascular growth, surrounded by endothelial cell masses. A thin network of fibrous tissue was present. Histologically the growth was indicative of a benign angioendothelioma of the palate."

However, in the desire to check this diagnosis, the slides were submitted to pathologists of the two medical schools in San Francisco. The report from the pathologist at Stanford University was as follows: "There was mucinous degeneration of the stroma, with an epithelial sweep of cells and some polyglandular arrangement. The diagnosis is that of a benign mixed tumor originating in salivary glandular tissue in the palate." A pathologist at the University of California reported: "There were epithelial cells with hyalinized connective tissue, which was punched throughout with holes. The growth is a simple benign mixed tumor of the parotid gland."

The weight of evidence was therefore preponderantly in favor of the diagnosis of a benign mixed tumor of the salivary gland.

Careful perusal of the literature pointed to surgical removal or radium treatment. Surgical therapy, in this case, was relegated to the background, for three reasons:

1. The tumor was histologically benign; it was of recent origin, and there was no involvement of glands.

2. Excision would leave a large defect in the palate, necessitating a plastic follow-up operation or a dental prosthesis.

3. Impairment of speech of greater or less degree would necessarily follow.

Accordingly, the patient was placed under a regimen of radium therapy, as follows: 50 mg. in rubber filter, 1 millicurie hour, on the following dates: April 27, May 11 and 29, June 19, July 13 and August 10; on September 18, he was given 40 mg. in rubber filter, 1 millicurie hour.

The patient has since been seen several times; the tumor mass has decreased notably in size, although it is a little firmer in consistency than before. There are no symptoms in connection with its presence, and the patient is in excellent spirits.

516 Sutter Street.

FIXATION OF THE CRICOARYTENOID JOINTS DUE TO STREPTOCOCCIC INFECTION

Improvement with the King Operation

MERVIN C. MYERSON, M.D., NEW YORK

The operation of Brien T. King, in which the omohyoid muscle is attached to the arytenoid cartilage, was devised for cases of bilateral abductor paralysis occurring after operation on the thyroid gland. Up to the present there is no record of this operation's having been performed for any other condition.

This report concerns a case in which the King operation was successfully performed on a man whose larynx was involved by a severe streptococcic infection. The infection caused fixation of both cricoarytenoid joints, so that almost complete closure of the glottis resulted. Tracheotomy became necessary. Five months later a King operation was performed.

REPORT OF A CASE

G. S., a 28 year old Negro, was admitted to the Kings County Hospital on Oct. 22, 1938. Six days before admission a peritonsillar abscess involving the right side of his throat had been incised. He came to the hospital because of bleeding from the region of the incision. Examination revealed nothing of importance except for the throat. A blood clot filled an incised wound of the right supratonsillar region of the palate. There was bleeding from this area, which was readily controlled by removal of the clot and the application of slight pressure. The patient remained in the hospital for five days and was then discharged as recovered.

Three days after he left the hospital, the patient became hoarse. His hoarseness continued up to the time of his readmission, on Nov. 20, 1939. He also experienced some difficulty in swallowing and had the sensation of a lump in his throat.

A week before his return to the hospital the patient had occasional spells of shortness of breath, even while at rest. As time passed he experienced considerable dyspnea during both inspiration and expiration. Examination of his larynx disclosed extensive edema of all the structures of the right side. The tissues were bright red, an indication of active inflammation. The right vocal cord could not be visualized. The left arytenoid eminence was slightly swollen and red; the left vocal band could be seen in its entirety. The glottic space was greatly reduced.

An abscess of the right side of the larynx was suspected, but its presence could not be confirmed by direct laryngoscopic examination. A low tracheotomy was performed. Within a few days the acute inflammation subsided. The larynx was not as red as before, but there were still considerable swelling of the right arytenoid area and slight swelling of the left. The right vocal cord had become fixed, and there was only slight motion of the left. Three months after tracheotomy little swelling remained on the right side; the left arytenoid area was no longer swollen. By that time, however, there was no motion of either cord. Both cricoarytenoid joints were fixed, and the vocal cords were closely approximated in the midline position.

In February, three months after tracheotomy, it occurred to me that manipulation of the cricoarytenoid joints might mobilize the arytenoid cartilages and the attached cords and overcome the laryngeal stenosis. A laryngofissure was performed, and both joints were manipulated digitally. There was no local reaction as a result of this procedure, but the right arytenoid cartilage was moved slightly anterior to the left. The arytenoid cartilages appeared more closely approximated than before; the vocal cords remained in a position of almost complete adduction.

Five weeks after the unsuccessful manipulation of the arytenoid cartilages a King operation was performed on the right side, with local anesthesia. The operative procedure was greatly facilitated by the unusually large size of the individual structures. An incision was made along the anterior border of the sternocleidomastoid muscle. Dissection was carried through the skin, the superficial fascia and the platysma muscle, down to the superficial layer of the deep fascia. The omohyoid muscle was identified, as were also the sternohyoid and

thyrohyoid muscles. The omohyoid was freed and severed high up, near the hyoid bone. The contents of the carotid sheath were retracted laterally. The larynx was rotated so as to bring its posterior aspect into the wound. The fibers of the inferior constrictor muscle were divided, and part of the right cricoarytenoideus posticus muscle was freed in order to expose the arytenoid cartilage. The attachments of the interarytenoideus, cricoarytenoideus posticus and lateralis muscles to the arytenoid cartilage were severed. The ligamentous attachments of the cricoarytenoid joint were cut with a pair of small, curved, sharp scissors. The loose end of the omohyoid muscle was sutured to the right arytenoid cartilage. This was accomplished with difficulty; twice the passage of the needle through the tip of the muscular process fractured the cartilage. The arytenoid cartilage was not sutured to the ala of the thyroid cartilage, as advocated by King, because it was my impression that this step could be omitted in my case.

The postoperative course was uneventful.

Frequent observations were made on the larynx and the patient's capacity to carry on without the aid of the tracheal cannula.

In the latter part of July 1941, almost two and a half years after the King operation was performed, the patient presented himself for decannulation. At this time he stated that he had worn a cork in his tracheotomy tube since June 1939, three months after his operation. Only recently had he decided to have the tube removed, because its retention no longer served his purpose. He had been suing his employer for damages because of the throat condition he had contracted in October 1938. His job consisted of emptying bags of cement into a mixer. He contended that exposure to the cement dust had caused the original ailment of the throat, and he attributed his incapacity to his work. In order to strengthen his case, he claimed that he could not get along without the tracheotomy tube. Whenever he was examined, he failed to mention that he had been wearing a cork. At each examination it was concluded that he did not have sufficient glottic space to permit removal of the tube. All attempts to cork the tube failed because of his lack of cooperation. Shortly before his last admission to the hospital the patient lost his lawsuit, and with it the incentive to continued malingering.

We who examined him in the follow-up clinic, being unaware of the legal aspects of the case, ascribed the patient's reluctance to having the tube removed to a psychologic reaction. After decannulation it was found that he could not carry on with the hard labor to which he had been accustomed. He could, however, comfortably undertake normal physical activity.

At the time of decannulation, and subsequently, there were retraction of the right vocal cord outward and a glottic space approximately two thirds of normal. During phonation the left vocal cord appeared to move, while the right cord was at rest.

The King operation was performed on an ankylosed cricoarytenoid joint, which resulted from a streptococcic infection. Even though the arytenoid cartilage was not pulled outward by a ligature between it and the thyroid cartilage, the end result was good. The operation was carried out four months after a tracheotomy was performed. The patient was under observation during this interval, a fact which would eliminate the possibility of there having been some degree of recovery of the joint.

CONCLUSIONS

Mobilization of a cricoarytenoid joint fixed by a severe inflammatory reaction could not be accomplished by digital manipulation through a laryngofissure exposure.

The King operation appears to be of value in the treatment of laryngeal stenosis caused by fixation of both cricoarytenoid joints as a result of streptococcic infection. Fixation of these joints resulting from tuberculous infection is a different problem and will not permit manipulation; such manipulation would activate the tuberculous process.

136 East Sixty-Fourth Street.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

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PLASTIC SURGERY FOR 1942

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The literature on plastic surgery for 1942 shows a stimulated interest in the management of facial injuries and a wider application of the sulfonamide compounds in the local therapy of wounds. There are also many contributions on the treatment of shock and the problem of improving the general condition of injured patients before skin grafting or other surgical procedures are undertaken.

These trends demonstrate that the otolaryngologist doing facial plastic surgery must be trained to give satisfactory early treatment for acute injuries of the face and to understand the general replacement therapy and dietary regimen for patients in shock.

An important contribution in 1941 was the experimental work of Dupertuis,[1] which demonstrated the actual growth of young auricular and costal cartilage grafts in rabbits. If young human cartilage grafts continue to grow after transplantation, the surgical handling of saddle nose and other deformities in children may require revision.

In this review the excellent outline previously used by Ivy and Miller is followed, with the addition of illustrations of interesting procedures and occasional editorial comment on articles dealing with controversial or new concepts.

RESULTS OF CLINICAL AND LABORATORY RESEARCH—GENERAL PRINCIPLES

LOCAL THERAPY WITH SULFONAMIDE COMPOUNDS

Lyons and Burbank,[2] in a collective review of local therapy with sulfonamide compounds, state that sulfanilamide is more soluble in serum than the other sulfonamide drugs. Sulfathiazole, sulfadiazine and sulfapyridine follow, in the order of their decreasing solubility. These facts may serve as a basis for selection of a particular drug or a combination of drugs for local application. The authors believe that the enthusiastic clinical adoption of local therapy with such compounds has failed to provide factual data which indicate the extent to which such treatment has prevented the growth of bacteria in wounds.

Bick[3] treated 50 patients with traumatic or operative wounds of the soft tissues by cleansing of the surface with soap and water and debridement when necessary. He then introduced 1.5 to 10 mg. of powdered sulfanilamide or sulfathiazole into the wound, covering all surfaces and suturing some of the wounds. A series of 50

1. Dupertuis, S. M.: Actual Growth of Young Cartilage Transplants in Rabbits: Experimental Studies, Arch. Surg. **43**:32 (July) 1941.
2. Lyons, C., and Burbank, C.: Local Sulfonamide Therapy, Surg., Gynec. & Obst. **74**:571, 1942.
3. Bick, E. M.: Observations on the Topical Use of Sulfonamide Derivatives, J. A. M. A. **118**:511 (Feb. 14) 1942.

wounds in which sulfonamide compounds had not been used served as controls. Comparison of the wounds in which such compounds were used with the controls indicated that the sulfonamide drugs retarded healing by at least 50 per cent and promoted excessive cutaneous scarring. He concluded that the introduction of sulfanilamide or sulfathiazole may interfere with the cosmetic result in plastic skin repair and that topical use of the drugs should be limited to cases in which infection is anticipated.

Taylor [4] advises against the prophylactic implantation of any of the sulfonamide compounds in a clean operative wound or in an infected wound which is to be closed. He warns that both sulfanilamide and sulfathiazole cause an inflammatory reaction when introduced into tissues in powdered form. Taylor inserted 1 grain (0.065 Gm.) of one of several common powdered sulfonamide compounds, which had been autoclaved, beneath the rectus sheath in dogs under aseptic conditions. Sections removed after three days showed that all the drugs produced inflammatory reactions, with severe edema and accumulation of inflammatory cells. When sulfathiazole and sulfadiazine were used, actual abscesses were formed about crystals which still persisted in the tissues. No crystals remained at the end of the three day period in animals in which any of the other drugs had been implanted (sulfanilamide, sulfapyridine and sulfaguanidine). Inflammatory changes were least with sulfanilamide.

[Comment: The successful take of skin grafts over infected areas to which sulfanilamide has been applied may be due to the milder type of inflammatory reaction produced by this drug. When one is grafting over infected granulations, finely powdered sulfanilamide is dusted lightly on the granulating surface and a skin graft applied over the area. In such cases the sulfanilamide increases the probabilities of a successful take and does not interfere with the nourishment of the graft. Sulfanilamide is more soluble in serum than are the other sulfonamide compounds and, for this reason, may be less irritating to human tissues.

Taylor's advice against the "prophylactic" implantation of the sulfonamide compounds in a clean operative wound seems rational. Such a procedure may give the surgeon a false sense of security, to the detriment of sound surgical judgment.]

Weil, Whitaker and Rusbridge [5] used sulfathiazole locally in the treatment of fresh compound wounds, compound fractures and simple fractures requiring operation. They reported that for the most part the wounds healed completely in a normal period, without undue accumulations of serum and without any unusual production of scar tissue. The authors advise complete debridement of all wounds before sulfathiazole is applied and the use of a high protein diet with vitamin C to promote tissue healing. They state that procaine inhibits the bacteriostatic action of sulfathiazole, and they advise against the use of local anesthesia in all cases in which sulfathiazole is to be used in prophylaxis against infection. Protein degeneration products from injured tissue also inhibit the action of sulfathiazole, and debridement of a wound is therefore important.

Horwitz [6] states that the local implantation of sulfanilamide crystals has no apparent adverse effect on the fate of transplanted bone. He bases this conclusion on experimental observations on 18 rabbits and a limited clinical experience with 5 patients.

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4. Taylor, F. W.: The Misuse of Sulfonamide Compounds, J. A. M. A. **118**:959 (March 21) 1942.

5. Weil, G. C.; Whitaker, D. W., and Rusbridge, H. W.: The Local Therapeutic Effect of Sulfathiazole, Am. J. Surg. **55**:374, 1942.

6. Horwitz, T.: The Effect of Sulfanilamide Crystals Used Topically on the Fate of Transplanted Bone, Surgery **11**:690, 1942.

MANAGEMENT OF BURNS

Penberthy and Weller [7] advise the intravenous administration of blood plasma and adrenal cortex extract for treatment of the shock accompanying burns. They remove all dead tissue from the burned area and apply 5 per cent tannic acid or some other coagulant. For the face and hands, however, they apply gauze wet in saline solution or impregnated with petrolatum and cover this with a firm pressure dressing to minimize edema and prevent loss of tissue fluids. They advise early skin grafting for deeply burned areas to prevent chronic infection and to lessen the patient's subsequent deformity.

[Comment: Adrenal cortex extract is valuable not only for shock accompanying burns but for postoperative shock. For this reason, it is a good routine measure to administer adrenal cortex extract to the patient before and after any extensive operative procedure. A high protein diet, reenforced with all the vitamins, should be given for three weeks before operation and an excess of sugar the day before operation.

The value of early skin grafting for third degree burns is now generally recognized. Severely burned patients are often worn out by three to four weeks of pain, and they are depleted by their loss of protein, fluid and mineral substances. A certain percentage of these severely burned persons will eventually die in spite of replacement therapy unless the extensive burned areas are given skin grafts.]

Allen and Koch [8] advise thorough cleansing of the burned area, followed by the application of gauze impregnated with petrolatum instead of a coagulant, such as tannic acid. Sterile dried gauze and mechanic's waste are laid over the petrolatum gauze, and the whole dressing is held in place with an elastic bandage to provide pressure. Allen and Koch stress the importance of pressure over a burned area as a means of preventing edema, or "white hemorrhage," and advise complete rest for the injured part. They remove the dressing at the end of twelve or fourteen days and apply skin grafts over all wide areas with loss of the whole thickness of skin.

Silers [9] uses the local treatment of Allen and Koch for burns and states that sterile compression dressings tend to prevent edema and hemoconcentration. Silers believes that patients are more comfortable with pressure dressings than with the use of coagulants, such as tannic acid.

Harkins [10] points out that the present conflict has produced types of burns not previously seen in large numbers, e. g., "airman's burn," the phosphorus burn and the burn contaminated with oil and sea water. When an explosion occurs in an airplane, the aviator throws up his hands to protect his face, incurring deep burns of both the hands and the face. Dressings soaked with saline solution or the local use of a sulfonamide compound, after thorough debridement, is recommended for a burn of this type. The phosphorus burn is apt to result from incendiary bombs and self-igniting phosphorus grenades. Small particles become embedded under the skin; such a burn should be immersed and the particles of phosphorus picked out under water. Successive washings with a mild solution of sodium bicarbonate,

7. Penberthy, G. C., and Weller, C. N.: Treatment of Burns, Surg., Gynec. & Obst. **74**:428, 1942.

8. Allen, H. S., and Koch, S. L.: The Treatment of Patients with Severe Burns, Surg., Gynec. & Obst. **74**:914, 1942.

9. Silers, V. E.: Primary Cleansing, Compression and Rest Treatment of Burns, Surg., Gynec. & Obst. **75**:161, 1942.

10. Harkins, H. M.: The Treatment of Burns in Wartime, J. A. M. A. **119**:385 (May 30) 1942.

1 per cent copper sulfate and boric acid should be followed by application of a dressing wet in saline solution. Oils and grease are to be avoided. In naval engagements burned patients are apt to be covered with grease or oil. Such substances may be removed with a grease solvent, like soap and water. The author emphasizes the importance of general treatment for shock, toxemia and sepsis and the advisability of early skin grafting.

Goldschlag [11] states that one of the most dangerous properties of mustard gas (dichlorethyl sulfide) is its great power of penetration. He advises the application of an ointment containing 0.2 per cent chloramine-T (sodium salt of paratoluene-*N*-chlorosulfonamide) and 8 per cent aerosol OT dry (dioctyl ester of sodium sulfosuccinate), which neutralizes the effect of the gas.

In discussing the management of injuries to soft tissues, Rank [12] states that as many as 70 per cent of the wounds in a ward for war casualties are infected with streptococci. The streptococcus often persists on what is thought to be a healthy wound, its presence inhibiting epithelial proliferation and preventing the take of skin grafts. The author advises the debridement of all wounds, removal of foreign bodies and application of sulfathiazole to the surface of the wound with a powder blower. Compresses wet in saline solution are then placed over the wound and held in place with gauze bandages.

Pickrell [13] reports on the use of prepared films of sulfonamide compounds in 50 cases of burn. The film contains sulfadiazine or sulfanilamide and is placed over the cleaned surface of the burn. A smooth, firm pressure dressing is then applied and the dressing changed in from three to five days.

GENERAL THERAPY

Starr [14] believes that vitamins are necessary for normal biologic processes and that a deficiency may manifest itself by serious changes in the body processes. He emphasizes the importance of nutritional disease (avitaminosis), which may be of critical significance in the patient's ability to survive the operation or to regenerate needed tissue after operation. In preparation for elective operations, he recommends the administration of a high protein diet and vitamin concentrates of all types for two or three weeks before operation. Additional vitamin C, vitamin K and the B complex vitamins should be given immediately before and for several days after all operations.

Hartzell and Stone [15] found that wound healing was retarded in guinea pigs kept on a subscurvy diet. In animals operated on while in a subscurvy state and given vitamin C in large doses after operation, the wounds reached the same degree of tensile strength as in normal animals by the eighth postoperative day. Histologic studies showed that the low tensile strength in wounds of partially vitamin C–deficient animals was due to a failure in production of collagen.

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11. Goldschlag, F.: Experiments on the Improvement of the Treatment of Mustard Gas Lesions of the Skin, M. J. Australia **1**:620, 1942.

12. Rank, B. K.: Base Hospital Management of Soft Tissue Injuries, Australian & New Zealand J. Surg. **11**:171, 1942.

13. Pickrell, K. L.: A Sulfonamide Film for Use as a Surgical Dressing, Bull. Johns Hopkins Hosp. **71**:304, 1942.

14. Starr, P.: The Value of Vitamins in Surgical Practice: Collective Review, Surg., Gynec. & Obst. **74**:309, 1942.

15. Hartzell, J. B., and Stone, W. E.: The Relationship of the Concentration of Ascorbic Acid of the Blood to the Tensile Strength of Wounds in Animals, Surg., Gynec. & Obst. **75**:1, 1942.

Bartlett, Jones and Ryan [16] conclude that a depletion of vitamin C produces a decreased ascorbic acid content and a decreased tensile strength in healing wounds of the fascia and skin in human beings. In spite of a low ascorbic acid level in the plasma at the time of operation, normal wound healing may be produced by adequate vitamin C therapy during the postoperative period.

Mulholland [17] states that knowledge of the functions of the liver is necessary for the understanding of many principles involved in preoperative care. There is definite evidence that the liver can be fortified against injury or impairment of its functions by specific measures. Mulholland advises the preoperative administration of a high protein diet to promote the removal of fat from the liver, since a fatty liver is susceptible to damage from anesthesia. The author emphasizes that a normal protein level in the plasma is necessary for normal wound healing and for prevention of shock.

SKIN GRAFTING

Smith,[18] in a plea for simplification of plastic procedures on the face, advises against the use of skin flaps or of free skin grafts except as measures of necessity. He contends that many types of facial disfigurement can be repaired by multiple excision and the Z plastic procedure, without the transfer of skin from elsewhere on the body. Such transfers create additional scarring in the area where the skin graft is removed, and the grafted skin on the face is often conspicuous and unsightly. The author supports his statements by photographs of persons who have had skin grafts from elsewhere on the body surface transferred to the face.

[Comment: Smith's paper is timely and represents an attempt to alter the present surgical approach to the problem of facial repair. The essence of the author's advice is to use local procedures, such as excision and rearrangement of the adjacent skin, whenever possible. One should resort to skin grafting only when the character or the extent of the deformity precludes successful correction by local measures.]

Brown and McDowell [19] advise the transplantation of thick split grafts as a covering for extensive third degree burns. The authors believe that the thick split graft is superior to the Ollier-Thiersch graft because the former has a pad of derma beneath the epithelium. Biopsies of a thick split graft demonstrate that the derma survives after transplantation and that the overlying epithelium is normal in every respect.

Brown and McDowell [20] summarize the procedures that have been found of value in repair of burns. They state that the healing of burned areas depends largely on the depth of destruction of skin and that the healing time and scarring are not greatly altered by the particular preparation used on the burned area.

[Comment: Careful observers will agree with Brown and McDowell that local therapy of the burned area does not greatly alter the rapidity of healing or the amount of scar contracture. Secondary anemia, hemoconcentration and depletion

16. Bartlett, M. K.; Jones, C. M., and Ryan, A. E.: Experimental Wounds in Guinea Pigs and Ascorbic Acid Content and Tensile Strength of Healing Wounds in Human Beings, New England J. Med. **226**:469, 1942.

17. Mulholland, J. H.: The Role of the Liver in Preoperative Care, Ann. Surg. **115**:148, 1942.

18. Smith, F.: Some Refinements in Reconstructive Surgery of the Face, J. A. M. A. **120**:352 (Oct. 3) 1942.

19. Brown, J. B., and McDowell, F.: Epithelial Healing and the Transplantation of Skin, Ann. Surg. **115**:1166, 1942.

20. Brown, J. B., and McDowell, F.: Massive Repairs of Burns with Thick Split Grafts, Ann. Surg. **115**:658, 1942.

of the plasma protein profoundly retard the growth of skin in the burned area and the take of skin grafts applied to the burned area. All the factors influencing epithelial regeneration and the take of skin grafts are not known, and occasionally one will make a graft on a patient who is in good condition from the "laboratory standpoint" but who seems to have a sort of allergy to his own transplanted skin.]

Young [21] advises the immediate cleansing and complete debridement of a burn, with immediate skin grafting of deeply burned areas.

Fig. 1.—*A*, ectropion, cataract, atrophy of the skin and underlying tissues, arrested development of bone and partial destruction of the lip as the result of roentgen irradiation of a pigmented nevus in early childhood.

B, reconstruction of the ectopic eyelid with a full thickness skin graft from the upper lid, showing surgical adhesions of the lids to prevent contraction during the period of organization and outline of the flaps for reconstruction of the lip and face.

C, completed reconstruction of the lip; surgical adhesions of the margins of the lids and advancement of normal skin into the atrophic area.

D, appearance two months after completion of the repair.

From Smith,[18] figure 11.

21. Young, F.: Immediate Skin Grafting in the Treatment of Burns, Ann. Surg. **116**:445, 1942.

In discussing the treatment of war wounds, Converse [22] recommends total debridement of the wound, followed by the application of sulfanilamide powder over the surface of the wound. A skin graft is then applied and immobilized with a pressure dressing. Sulfathiazole was found to be less desirable than sulfanilamide because the former had a tendency to cake on the wound. Sulfanilamide dusted on the surface of the wound before application of the skin graft did not prevent the take of the graft.

For the correction of defects of the face, Maliniac [23] prefers the full thickness skin graft rather than the split graft. The latter takes easily and quickly, but its color is not always good, and it tends to contract. Tube and pedicle flaps are indicated when there is loss of skin and subcutaneous tissue, when lining and covering must be provided for defects involving the nose, cheek or lip and when the blood supply is poor or bone and cartilage are exposed.

Byars [24] states that the advantages of the full thickness skin graft over the split graft are that the former undergoes only slight contracture and no wrinkling and provides a covering which has a better appearance and texture. The disadvantages of the full thickness graft are as follows:

1. Uniform takes are more difficult to secure.

2. Removal of the graft creates an open wound at the donor site, which does not heal spontaneously by regeneration of skin.

3. The graft cannot be applied to ulcerating wounds with any degree of confidence.

4. The graft is slow and tedious to remove from its donor site and is tedious to apply.

Both full thickness and split skin grafts sometimes undergo excessive pigmentation after transplantation.

Dorrance and Bransfield [25] state that whenever the pericranium of the skull is removed surgically or by trauma, the defect should be covered immediately with a full thickness pedicle flap of adjacent scalp. The pedicle flap is raised from, but does not include, the pericranium. The defect created by the shifting of the pedicle flap has the pericranium for a floor, and it may be covered by a free graft taken from the abdomen or the thigh.

Straatsma,[26] in an unpublished address, emphasized the importance of early skin grafting for all types of war injuries. He stated that the Padgett dermatome is of great value in obtaining large sheets of skin from difficult donor sites, such as the abdomen and chest.

NOSE

Davis [27] discusses the general indications for plastic operations on the nose and warns that public interest in this subject has been unduly aroused by ill advised articles in lay magazines and metropolitan newspapers. Davis states, however, that real nasal deformities are numerous and that when operation is performed by

22. Converse, J. M.: Early Skin Grafting in War Wounds, Ann. Surg. **115**:321, 1942.
23. Maliniac, J. W.: Free Skin Grafts Versus Flaps in Surface Defects of Face and Neck, Am. J. Surg. **58**:100, 1942.
24. Byars, L. T.: Free Full Thickness Skin Grafts, Surg., Gynec. & Obst. **75**:8, 1942.
25. Dorrance, G. M., and Bransfield, W. J.: Immediate Covering of Denuded Area of Skull, Am. J. Surg. **58**:236, 1942.
26. Straatsma, C. R.: Unpublished address before the American Society of Plastic and Reconstructive Surgery, Baltimore, 1942.
27. Davis, W. B.: External Nasal Deformities and Methods Used in Their Repair, Arch. Otolaryng. **36**:619 (Nov.) 1942.

a properly trained plastic surgeon, the functional and cosmetic results are usually gratifying. The author believes that congenital deficiencies of the bony bridge are preferably corrected at the age of 17 to 20 years by autogenous rib cartilage transplants. When correction is attempted in childhood, the cartilage of course does not increase in size; consequently, as maturity is approached, there is an increasing disproportion between the size of the transplanted cartilage and the size of the developing nose. If, in rare cases, early correction is deemed advisable because of the pronounced introspection or some other psychologic factor, the parents should be told that a second operation will probably be necessary during late adolescence.

[Comment: Davis warns that an autogenous cartilage graft inserted to support a child's nose will be inadequate after the adolescent growth of the nose. This contention, which is generally accepted, means that an autogenous cartilage graft survives transplantation as living cartilage but does not increase in size after transplantation. Young cartilage is assumed to lose the power of growth after transplantation. Dupertuis, however, has demonstrated the actual growth of young auricular and costal cartilage grafts in rabbits. If young human cartilage grafts continue to grow after transplantation, as do young rabbit cartilage grafts, the management of saddle nose and other deformities in children may require revision. It is important, therefore, for some investigator to determine the presence or absence of actual growth in young human cartilage grafts.]

Fleming [28] prefers ivory for the filling in of defects of the nasal dorsum but states that bone or rib cartilage may be used. The ivory is introduced through an endonasal incision at the junction of the alar and the quadrilateral cartilage.

[Comment: At one time many plastic surgeons used ivory as a filling substance for saddle depressions of the nose. Observations over long periods in cases in which this substance was employed has induced most surgeons to discard the use of ivory and to substitute living autogenous cartilage or bone as a grafting material because the latter substances are more reliable in a larger percentage of cases. Preserved cadaver cartilage, as advocated by Pierce and O'Connor, is a desirable substitute for the living autogenous graft when good surgical judgment precludes removal of the patient's own cartilage or bone.]

MacCollum [29] states that he has found an autogenous bone graft from the ilium preferable to an autogenous cartilage graft for elevation of a depressed nasal bridge. Technical details for obtaining and preparing of the bone graft are given.

In the experience of Straith,[30] most injuries to the nose and face result from automobile accidents. For defects on the nose, the author prefers to use a graft from the upper eyelid for small deficiencies and skin from behind the ear for larger ones. In cases of fracture of the bones of the nose the fragments are replaced in their proper position to avoid subsequent deformity. Subcuticular sutures of nylon are used for lacerations of the skin to avoid postoperative stitch marks. If saddle nose results, in spite of all efforts to "hold up" the nasal bones, the depression is built up with rib cartilage. The author has used rib cartilage preserved in aqueous solution of merthiolate for this purpose. Some patients show reactions to preserved cartilage, but if such a reaction can be avoided, the risk of the operation on the chest is eliminated.

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28. Fleming, P. N.: The Saddle Nose, Dis. Eye, Ear, Nose & Throat **2**:244, 1942.
29. MacCollum, D. W.: The Elevation of the Nasal Bridge Line, Surgery **12**:97, 1942.
30. Straith, C. L.: Injuries and Deformities of the Nose, Tr. Indiana Acad. Ophth. & Otolaryng. **26**:88, 1942.

I [31] emphasize the difficulty of diagnosis of acute depressed fractures of the nose in infants and small children. In doubtful cases I advise manipulation of the nose with the patient under ether anesthesia. Special operative procedures for the correction of recent and old depressed fractures of the septal cartilage are described and illustrated. Displacement of the lower anterior border of the septal cartilage is repaired by complete exposure of the displaced cartilage, removal of a small portion of cartilage posterior to the displaced free border and resetting of the displaced segment in proper position between the folds of skin of the columella. I advise the Pierce technic for total reconstruction of the ear and the operation of Davis and Kitlowski for the correction of abnormally prominent ears. I state that operations for improvement of the hearing in cases of congenital meatal atresia are rarely successful.

King [32] advises the use of fine silk or horse hair sutures for the repair of recent wounds of the nose and face. When the advisability of operation on an old fractured nose is under consideration, the author warns that the indications must be definite and that there must be a "fair chance" for improvement.

[Comment: King's admonition to avoid operation on an old fracture of the nose without definite indication that the postoperative result will be satisfactory is important. It is both honest and wise to discuss the probable postoperative appearance of the nose with the patient. If the patient is not satisfied with this degree of improvement, the surgeon's best policy is to advise against operation. The desirability of improvement in the appearance of a nose depends largely on the patient's psychic makeup and, accordingly, varies widely for the same type of deformity among different patients. Many persons with minor nasal abnormalities should be advised against operation, since all surgical procedures are associated with some risk.]

Maliniac [33] lists the indications for plastic operations on the nose as dependent on two factors: (1) the mentality of the patient and his reaction to the deformity and (2) the extent of the malformation and the importance of its correction to the health and economic life of the patient. The author believes that a mental reaction to the deformity "out of all proportion" to the conspicuousness of the defect is an indication for psychiatric study of the patient.

McKenzie [34] includes among deformities causing obstruction at the nasal orifices the following conditions: curling or crushing of the anterior edge of the septal cartilage; deflection of the anterior edge of this cartilage; deflection, thickening or elongation of the columella; eversion of the medial crura of the alar cartilages; collapse of the lateral crura of the alar and triangular cartilages, and congenital atresia of the nares.

Weaver [35] reports 9 cases in which a rhinoplastic operation was performed. A saw was usually employed to remove the nasal hump and a fine rasp to smooth the edges after removal of the hump. The nose was refractured in all cases by sawing through the nasal process of the superior maxilla.

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31. Peer, L. A: Management of Deformities of the Nose, Ear, Eyelids, Lips and Palate in Infants and Small Children, Dis. Eye, Ear, Nose & Throat **2**:166, 1942.

32. King, E.: Plastic Surgery of the Nose and Face, Kentucky M. J. **40**:264, 1942.

33. Maliniac, J. W.: Plastic Surgery in Nasal Deformities, Dis. Eye, Ear, Nose & Throat **2**:102, 1942.

34. McKenzie, W. R.: The Nasal Entrance, South. M. J. **35**:433, 1942.

35. Weaver, D. F.: Plastic Surgery of the Nose, J. Michigan M. Soc. **41**:229, 1942.

Cinelli [36] concludes that downward trauma over the nose often results in displacement of the septal and lateral cartilages, so that nasal obstruction results. He describes the operative procedures used for the repair in 3 cases in which the cosmetic factor was of little concern to the patient but plastic repair was nevertheless indicated to relieve the symptoms of obstruction.

Greeley [37] advises early treatment for nasal fractures, with manipulative correction and immobilization of the displaced fragments. If the fracture has healed without correction, open reduction is necessary. Keloids are treated by a combination of irradiation and surgical intervention.

Waldron [38] believes that the immediate treatment of the injured nose should be directed toward the control of hemorrhage and the prevention of shock. Lacerated margins of the skin require conservative debridement, but partially separated fragments of bone and cartilage should not be removed. "Primary treatment of fractures of the nose may be undertaken within the first two or three weeks after injury." Treatment consists of combined external manipulation with the finger and intranasal elevation of the parts with a blunt elevator. The fragments are held in place by intranasal packing of ½ inch (1.27 cm.) lubricated iodoform gauze.

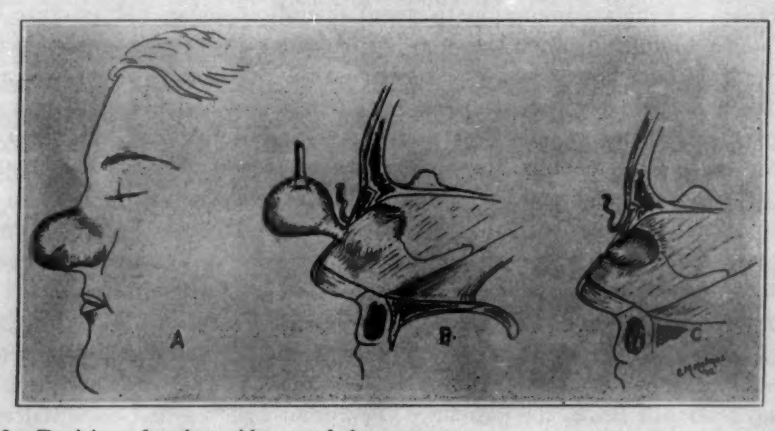

Fig. 2.—Excision of a dermoid cyst of the nose.

A, size and location of the cyst, with line indicating incision made for exposure of the cyst. *B*, schematic drawing, showing elevation of the main part of the cyst. The stalk, which penetrated into the septum beneath the nasal bones, is shown as a cystic swelling between the flaps of septal mucosa. *C*, excision of the cyst, including as much as possible of the portion extending into the septum. The cavity is left open into the nose to prevent possible recurrence of the cyst. From Holmes,[40] figure 3.

[Comment: If the general condition of the patient is satisfactory, acute fracture of the nose should be immediately reduced. The displaced bony fragments heal rather rapidly in their distorted position, and the satisfactory reduction of these fragments two weeks after injury may not be possible.]

Salinger [39] reports a case in which an extensive cavernous hemangioma involved the nose, the nasal septum and the forehead. After preliminary treatment with

36. Cinelli, A. A.: High Cartilaginous Septal Trauma, Dis. Eye, Ear, Nose & Throat **2**:6, 1942.

37. Greeley, P. W.: Nasal Deformities, Abrasions and Accidental Tattoos, Scars, Keloids and Scar Contractures, S. Clin. North America **22**:253, 1942.

38. Waldron, C. W: Injuries to the Nose, Minnesota Med. **25**:258, 1942.

39. Salinger, S.: Cavernous Hemangioma of Nose, Nasal Septum, and Forehead, Ann. Otol., Rhin. & Laryng. **51**:268, 1942.

radium needles, he excised the remaining portions of the growth and later removed the scar. The author believes that the preliminary irradiation materially reduced the vascularity of the tumor and thus made later surgical excision possible.

Holmes [40] reviews 15 cases of dermoid cyst of the nose operated on at the Massachusetts Eye and Ear Infirmary in the past twenty years. He describes the operative technic which he and Kazanjian used in 3 of the recent cases. Holmes states that dermoid cyst of the nose is not as rare as the literature indicates.

In commenting on the ideal nose, Cinelli [41] notes that measurement of the nose directly after operation does not allow for the swelling and edema that are present at the time and for the postoperative contracture which occurs later.

Cohen [42] advises against the doing of nasal plastic procedures in the presence of acute sinusitis, infected teeth or similar conditions. The author states that special technic is necessary in avoiding a low nasal hump after operation and, also, in dealing with the nasal tip, where errors of technic are most apt to occur.

Lewin [43] reported a case of anhidrotic ectodermal dysplasia and reviewed the literature. He repaired the retracted nose with a skin flap from the neck and inserted preserved cadaver rib cartilage to provide structural support for the nose.

FACE AND JAWS

Ivy [44] contributes an excellent article on the early and late treatment of the face and jaws as applied to war injuries. He emphasizes that the most efficient treatment of such casualties entails close cooperation of the officers of the Medical and the Dental Corps. In the combat area the following points demand special attention: (1) arrest of hemorrhage; (2) provision of an adequate respiratory airway; (3) temporary approximate reduction and fixation of bone fragments; (4) provision for dependent drainage, and (5) provision for safe transportation from the combat zone to the hospital in the rear.

Ivy states that treatment of burns of the hands and face with tannic acid has given unfortunate results. Application of an aqueous emulsion of sulfadiazine or boric acid ointment, a covering of fine mesh gauze and a pressure dressing is recommended as a first aid measure for the hand; for the face no dressing at all is recommended. Later treatment consists in cleaning of the surface with soap and water, careful irrigation and debridement of the blisters; then dressings similar to those described are applied. Ivy emphasizes that third degree burns should have skin grafts as soon as the granulating surface is clean.

Dorrance and Loudenslager [45] made the diagnosis of hypermotility of the upper lip when the patient in repose exposes the maxillary incisors and canines. When such a patient laughs, the upper teeth and the alveolar ridge above the teeth are exposed by excessive elevation of the upper lip. The chief factors in the deformity are shortening and overactivity of the muscles which elevate the upper lip. The authors describe their operative correction, which consists of division of the muscles which elevate the upper lip. Their approach is through an incision in the labio-dental fold.

40. Holmes, E. M.: Dermoid Cysts of the Nose, Ann. Otol., Rhin. & Laryng. **51**:662, 1942.
41. Cinelli, A. A.: The Ideal Nose, New York State J. Med. **42**:64, 1942.
42. Cohen, S.: Complications of Nasal Plastic Surgery, Dis. Eye, Ear, Nose & Throat **2**:235, 1942.
43. Lewin, M. L.: Nasal Deformity in Anhidrotic Ectodermal Dysplasia, Arch. Otolaryng. **35**:210 (Feb.) 1942.
44. Ivy, R. H.: Early and Late Treatment of the Face and Jaws as Applied to War Injuries, South. Surgeon **11**:366, 1942.
45. Dorrance, G. M., and Loudenslager, P. E.: Hypermotility of the Upper Lip, Surg., Gynec. & Obst. **75**:790, 1942.

Converse [46] discusses his experiences with war injuries of the face and advises primary suture of all lacerations involving the soft parts. When the edges of the wound cannot be approximated because of loss of tissue, a primary skin graft is indicated. By this means healing of the wound will result, and secondary plastic repair can be carried out later.

Metz [47] describes the surgical handling of facial wounds, malar-zygomatic fractures and burns. Marcks [48] presents the local and general considerations in the soft tissue repair of injuries about the face and head. In a discussion of fractures of the face, Naftzger [49] states that the facial wall of the maxilla and the

Fig. 3.—Hypermotility of the upper lip. *A*, face in repose, showing a short upper lip, with exposure of the incisor and canine teeth. *B*, patient smiling. The teeth and the alveolar ridge are exposed. From Dorrance and Loudenslager,[45] figure 1.

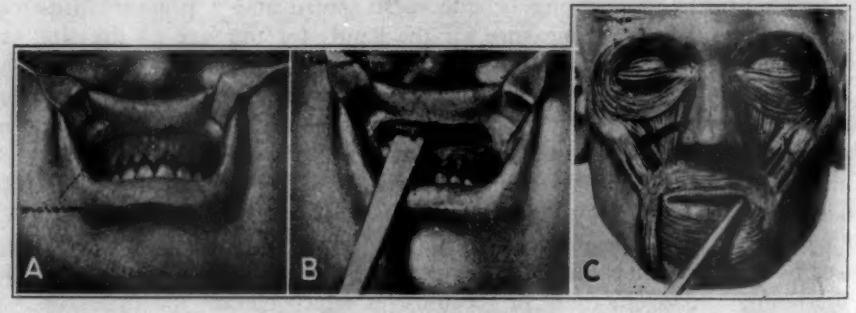

Fig. 4.—Surgical correction for hypermotility of the upper lip.
A, line of incision in the labiodental fold. *B*, elevation of the soft tissues from the maxilla with a periosteal separator. *C*, drawing showing division of muscles which elevate the upper lip. From Dorrance and Loudenslager,[45] figure 2.

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46. Converse, J. M.: War Injuries of the Face, Tr. Am. Acad. Ophth. (1941) **46**:250, 1942.

47. Metz, W. R.: Some Fundamentals of Plastic Repair, South. Surgeon **11**:502, 1942.

48. Marcks, K. M.: Soft Tissue Repair in Injuries About the Face and Head, Pennsylvania M. J. **45**:801, 1942.

49. Naftzger, J. B.: Fractures of the Face Involving Nasal Accessory Sinuses, Ann. Otol., Rhin. & Laryng. **51**:414, 1942.

orbital floor are usually fractured with blows over the malar eminence. If the orbital floor is depressed, there may be downward displacement of the eye, with consequent double vision. Elevation of the depressed bony floor of the orbit in cases of such injury requires exposure through an incision under the lip, as in a radical operation on the antrum. The author advises thorough cleansing of the wound, excision of badly lacerated tissues and removal of all foreign bodies. The edges of the wound are then sutured, and if the patient's condition permits, immediate reduction of the fracture is advised. A plaster head cast is essential in

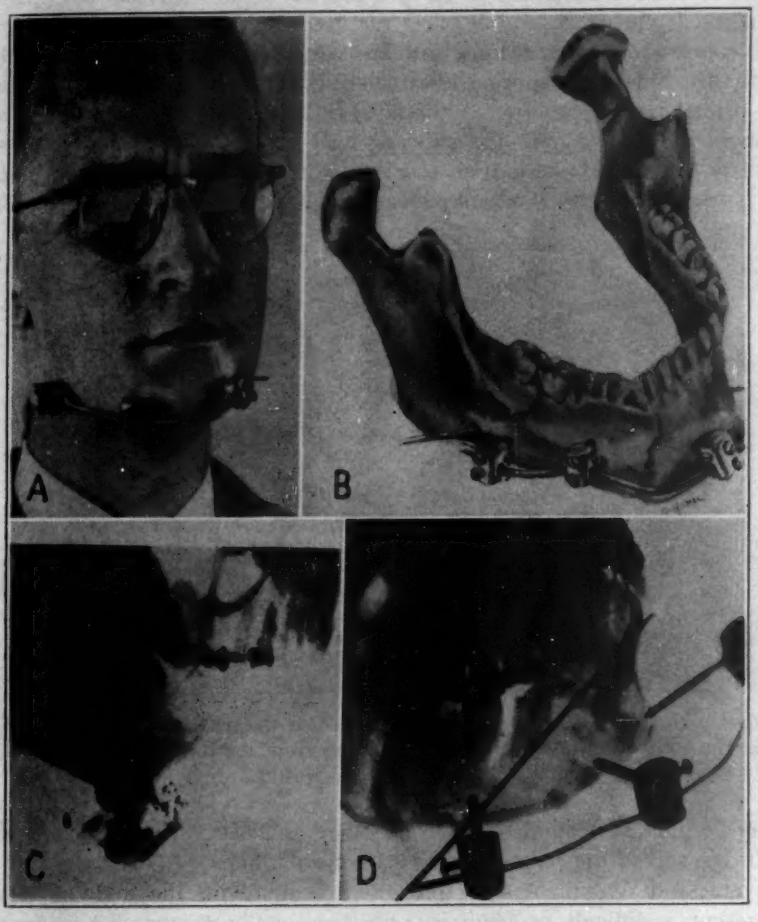

Fig. 5.—Reduction of fracture through the horizontal ramus and condyle of the mandible. All major fragments were saved, reduced and immobilized with the external bar and the internal wire. A few interdental wires were also placed where teeth were present for repair of the fracture of the condyle.

A, external bar, with screws and wire in position.

B, diagram of fractured mandible, showing screws inserted into the fragments, with fixation of these fragments by the external bar. A wire is shown penetrating each horizontal ramus for additional support.

C, roentgenogram of the fractured mandible.

D, roentgenogram, showing screws in fragments, with fixation by the external bar. The wire through each horizontal ramus is also shown. From Brown and McDowell,[50] figure 9.

cases of extensive transverse fractures of the face to provide supports. Wires fastened to the depressed fragments are attached to the supports with elastic bands

to provide continuous traction. This continuous traction elevates the depressed bones into normal position.

Brown and McDowell [50] describe a method for the reduction and fixation of fracture of the lower jaw. In their procedure a steel wire of the Kirschner type is drilled across the fracture line, so that the fragments are held in position. Severe shattering injuries require the combined use of interdental wiring, arch bars, fixation with internal wires and external bars applied with screws. The last method consists of insertion of a screw through the outside into each of the larger fragments of bone and then attachment of all the screws to an external bar. Interdental wires are used when the condyles are fractured.

Adams [51] contributes a technic for the reduction and fixation of maxillary and facial fractures. For the approach two small incisions are made at the outer or the inferior orbital ridge. Steel wires threaded through small holes in the orbital ridge are brought downward over the anterior wall of the antrum and fastened to the teeth. The fractured bone is then elevated, and the wires are drawn taut. If the patient is edentulous, the wires may be attached to the false plate; otherwise, dental

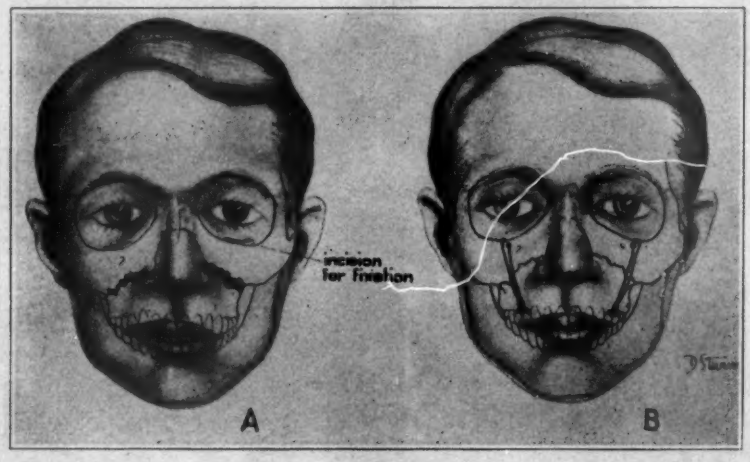

Fig. 6.—*A*, complete bilateral horizontal fracture of the maxilla. *B*, maxilla elevated and maintained in normal position with wires extending from the infraorbital ridges to the upper teeth. From Adams,[51] figure 1.

wax may be molded around the alveolar ridge and over the hard palate, the wires being drawn across the palate and tied in the midline.

Converse and Waknitz [52] state that fractures involving the angle of the mandible often present a difficult problem in reduction owing to the displacement of the posterior fragment. The authors have used an apparatus on the principle of the Roger Anderson pin unit to provide external fixation. The advantages of the method are that it allows accurate control of the fragments and permits the patient to open and close his mouth and to masticate. The disadvantage is the danger of infection.

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50. Brown, J. B. and McDowell, F.: Internal Wire Fixation of Jaw Fractures, Surg., Gynec. & Obst. **74**:227, 1942; **75**:361, 1942.

51. Adams, W. M.: Internal Wiring Fixation of Facial Fractures, Surgery **12**:525, 1942.

52. Converse, J. M., and Waknitz, F. W.: External Skeletal Fixation in Fractures of the Mandibular Angle, J. Bone & Joint Surg. **24**:154, 1942.

Rushton and Walker [53] treat mandibular fracture by drilling pins through the skin into the mandibular fragments. These pins are joined by bars, which permit rigid control of the fragments in every plane.

Berry [54] describes a method of reduction and fixation of the malposed fragments of a fractured mandible which is partially or totally edentulous. In cases of such a fracture interdental wiring cannot be used because of the absence of teeth. The appliance consists of special vitallium screws placed upright in the mandible on both sides of the fracture in the same position as the teeth. The screws are connected with an alinement rod, which may be bent and adapted to hold the fragments in the desired position.

In a discussion of the surgical correction of mandibular prognathism, Kitlowski [55] states that the condition can be corrected by section through the ramus

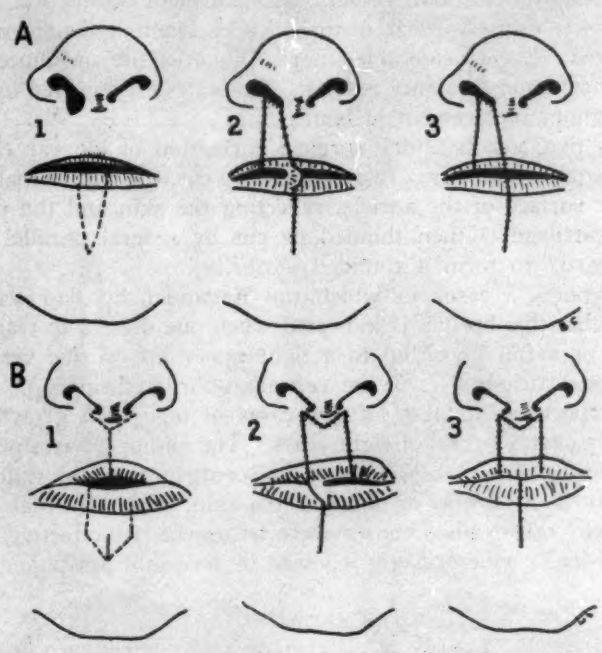

Fig. 7.—*A*, diagrams illustrating use of a vermilion-bordered flap for secondary correction of harelip.

1, tight retracted upper lip and deficiency in the floor of the nose. The condition followed an unsatisfactory operation for repair of unilateral harelip. The outline of the vermilion-bordered flap is indicated by dotted lines. *2*, flap sutured in place in the upper lip, with the pedicle crossing the mouth. *3*, completed repair, showing relaxation of the upper lip and correction of defect in the floor of the nose.

B, diagrams illustrating use of a split vermilion-bordered flap for secondary correction of a double harelip.

1, tight retracted upper lip, resulting from an unsatisfactory operative repair of a double harelip. The dotted lines indicate the outlines of the flap in the lower lip and the relaxing through and through incision in the upper lip. *2*, flap sutured in place in the upper lip, with the pedicle crossing the mouth. *3*, completed repair.

From Cannon,[60] figures 7 and 9.

53. Rushton, M. A., and Walker, F. A.: Mandibular Fractures Treated by Pin Fixation, Am. J. Orthodontics **28**:307, 1942.

54. Berry, H. C.: The Implantation Method of Setting Fractured Mandibles, Am. J. Orthodontics **28**:292, 1942.

55. Kitlowski, E. A.: The Surgical Correction of Mandibular Prognathism, Ann. Surg. **115**:647, 1942.

on each side. This procedure has the advantage that an opening is avoided into the mouth, but the objectionable feature that there may be interference with function because of the backward displacement necessary. Kitlowski advises removal of a section of bone from the body of the mandible for the correction of prognathism. He states that this procedure can be performed without danger of permanent loss of sensation or of the vitality of the teeth. A simple bridge attached to the teeth will hold the three sections of the mandible together satisfactorily.

Scher [56] advises osteotomy through the ascending ramus of the mandible for the correction of prognathism or of retruding lower jaw. Newman [57] also prefers section through the ascending ramus for the repair of prognathism.

According to Young,[58] the ascending or the horizontal ramus of the lower jaw, or both, can be removed without too serious impairment of function. If the region of the symphysis is excised, great disturbance in function results unless the loss of bone is repaired. If a considerable amount of soft tissue and mucosa is removed with the bone, functional disability results. This can be prevented by repair of the mucosal loss without replacement of bone.

Coe [59] states that a satisfactory surgical correction of lop ear entails the formation of an adequate antihelix. In repairing lop ear, the author makes an incision on the posterior surface of the auricle, reflecting the skin and the perichondrium. The auricular cartilage is then thinned or cut by several parallel incisions and "buckled backward" to form a rounded antihelix.

Cannon [60] reports 5 cases in which the Estlander lip flap was used. The author believes that this lip flap is indicated when one desires to transfer lip tissue from a normal or a full lower lip to a tight upper lip or vice versa. Excellent drawings illustrate various uses of the vermilion-bordered lip flap.

Conway [61] reports an analysis of 100 cases of malignant growths of the face treated surgically over a period of eight years. The author advises primary surgical excision and plastic repair in preference to roentgen ray and radium treatment.

Maliniac,[62] in a discussion of lupus of the skin, concludes that surgical intervention is the only resort when conservative treatment is ineffective. The surgical procedure of choice is wide excision followed by free skin grafting.

MISCELLANEOUS

Kazanjian [63] reports 3 cases of congenital choanal atresia, the condition being bilateral in 1 case and unilateral in 2 cases. Kazanjian approaches the atresia by incising through the base of the columella and displacing to one side a flap containing the columella and the septal cartilage. The exposed vomer, with its covering of mucous membrane, is then removed back to the choanae and the area of atresia removed laterally to the outer wall of the nose.

56. Scher, S. L.: The Deformed Chin and Lower Jaw, Ann. Surg. **115**:869, 1942.
57. Newman, J.: Repair of Prognathic and Retruded Jaws, Am. J. Surg. **58**:35, 1942.
58. Young, F.: Function of the Lower Jaw Following Partial Resection, Surgery **11**:966, 1942.
59. Coe, H. E.: Correction of Lop Ears, Northwest Med. **41**:126, 1942.
60. Cannon, B.: The Use of Vermilion-Bordered Flaps in Surgery About the Mouth, Surg., Gynec. & Obst. **74**:458, 1942.
61. Conway, H.: Principles of Plastic Surgery in the Treatment of Malignant Tumors of the Face, Surg., Gynec. & Obst. **74**:449, 1942.
62. Maliniac, J. W.: Surgical Treatment of Lupus of the Skin, Am. J. Surg. **55**:123, 1942.
63. Kazanjian, V. H.: The Treatment of Congenital Atresia of the Choanae, Ann. Otol., Rhin. & Laryng. **51**:704, 1942.

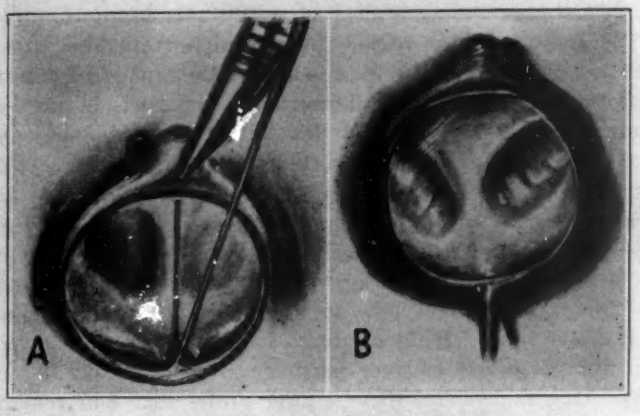

Fig. 8.—Choanal atresia. *A*, nasopharyngeal mirror image of unilateral choanal atresia. B, nasopharyngeal mirror image of bilateral choanal atresia. From Kazanjian,[63] figure 1.

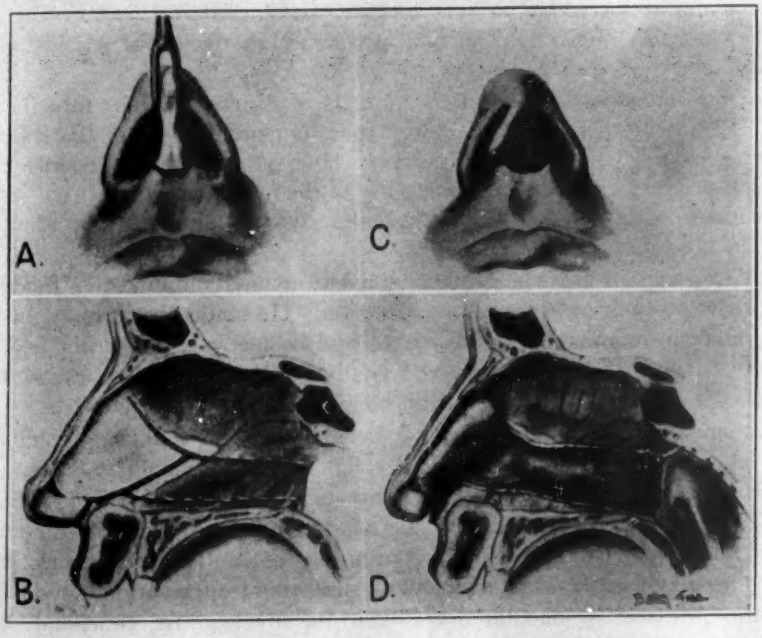

Fig. 9.—Surgical correction for choanal atresia. (*A*) The incision through the base of the columella is in three distinct planes, so that when the cut is resutured it will approximate exactly and leave a minimum amount of scarring. (*B*) The incision is carried along the floor of the nose, completely through the cartilaginous septum, back to the vomer, upward to the perpendicular plate of the ethmoid bone and then forward, with formation of a flap of the cartilaginous septum, which is displaced to one side. The dotted lines indicate the area where the vomer, with its covering mucous membrane, is removed back to the atresia. (*C*) The columella and septal cartilage flap are displaced to expose the vomer. (*D*) The septal cartilage flap is displaced to one side and the vomer, with its covering mucous membrane, is removed back to the atresia. The area of atresia has been removed laterally to the outer wall of the nose. The septal cartilage flap is then replaced and the columella sutured to the upper lip.
From Kazanjian,[63] figure 2.

Schwartz and Isaacs [64] report on 2 patients with congenital choanal atresia. One patient was successfully operated on, but the second died of carcinoma of the prostate, without operation for the atresia. An autopsy on the second patient demonstrated that the occluding wall was partly membranous and partly bony.

Pelliciari [65] gives a concise review of the history of the various types of cartilage grafts and the results of experimental studies made to determine the fate of cartilage grafts following transplantation. On the basis of the known clinical and experimental observations, he concludes that living autogenous cartilage is the best type of cartilage graft to use for the support of soft tissues.

[Comment: There appears to be considerable difference of opinion among plastic surgeons regarding the indications and use of the various cartilage grafts. A rather large group advocates the exclusive use of preserved cadaver cartilage, while another, more conservative, group uses only living autogenous cartilage. In the opinion of my associates in this department and myself, living autogenous cartilage is the first choice, living cartilage from another person (isogenous) is second choice and preserved cadaver cartilage is third choice. Young patients have a long life expectancy, and they require grafts that will remain throughout life. In such cases autogenous cartilage is the material of choice. Living autogenous cartilage grafts are also indicated in children because of the growth possibilities of young cartilage grafts. Preserved cadaver cartilage is a good substitute material for filling defects in older patients or for use in any case in which the patient's general condition precludes the removal of his own rib cartilage.]

Bothe and Davenport [66] made two implants of different metals into the femurs of cats. Examination after six to eleven months convinced them that electrolysis was not the primary cause of unfavorable bone reactions. According to these authors, the primary causes are the solubility of the metals and the degree of toxicity of the products of dissolution. They conclude that vitallium or tantalum are the probable metals of choice.

Beck [67] reported 4 cases in which he substituted vitallium plates for the skull bone which was removed in the same operation. He made observations in his first case for sixteen months and noted excellent cosmetic support and no clinical evidence of tissue reaction. The curved vitallium plates were placed close together and fastened to the margins of the skull bone with vitallium screws.

[Comment: The excellent work of Venable and his associates demonstrated that the alloy called vitallium, made of cobalt, chromium and molybdenum, is a nonelectrolyte, and hence does not give rise to tissue reaction. It remains to be proved, however, that tissues of the human scalp will tolerate vitallium for long periods. The clinical surgeon should continue to use autogenous bone or cartilage grafts for repair of depressions in the skull until more information is available concerning the ultimate fate of vitallium. From the investigative viewpoint, Beck has contributed a valuable article.]

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64. Schwartz, A. A., and Isaacs, H. J.: Congenital Atresia of Posterior Nares, Arch. Otolaryng. **35**:603 (April) 1942.

65. Pelliciari, D.: Use of Cartilage Transplants in the Reconstruction of the Facial Architecture, Mil. Surgeon **91**:324, 1942.

66. Bothe, R. T., and Davenport, H. A.: Reaction of Bone to Metals, Surg., Gynec. & Obst. **74**:231, 1942.

67. Beck, C. S.: Repair of Defects in Skull by Ready Made Vitallium Plates, J. A. M. A. **118**:798 (March 7) 1942.

For the treatment of hemangiomas, Bailey and Kiskadden [68] believe that application of the gamma rays of radium, in the form of well filtered packs, give more uniform results than roentgen therapy. The authors classify hemangiomas at considerable length, for they believe that the histologic structure of the growth should determine the method of therapy.

Davis and Stafford [69] describe an interesting technic for the construction of the extrathoracic esophagus. The authors present a case in which this technic was successfully used, and the article contains drawings clearly illustrating the operative steps of the reconstruction.

Holt and Falls [70] describe the technic for making latex negatives, from which positives can be formed. They list the numerous advantages of the latex mold over other types. Brown [71] also prefers the latex mold, and he describes his technic for constructing latex prostheses to substitute for the ear and nose and to repair orbital defects.

965 Broad Street.

68. Bailey, W., and Kiskadden, W. S.: The Treatment of Hemangiomas with Special Reference to Unsatisfactory Results, Radiology **38**:552, 1942.

69. Davis, J. S., and Stafford, E. S.: Successful Construction of an Extrathoracic Esophagus, Bull. Johns Hopkins Hosp. **71**:191, 1942.

70. Holt, C. S., and Falls, F. H.: The Use of Latex and Hydrocal in Casting Medical Sculpture, Surg., Gynec. & Obst. **74**:1133, 1942.

71. Brown, A. M.: Correction of Facial Defects with Latex Prostheses, Arch. Otolaryng. **35**:720 (May) 1942.

Abstracts from Current Literature

Ear

A Radiologic Study of Mastoids in Children. L. A. Gagnier Jr., Canad. M. A. J. **45**:312 (Oct.) 1941.

The roentgenologic study of the mastoid of a child from birth to 5 years of age presents a certain difficulty of interpretation and is limited particularly to the antral and periantral regions.

However, during this period the examination is of value.

Moore, Omaha [Am. J. Dis. Child.].

A Suggestion for a New Method of Testing Hearing in Aviation Candidates. D. B. Fry, J. Laryng. & Otol. **57**:11 (Jan.) 1942.

Fry considers it generally agreed that the usual hearing tests for the selection of flying personnel are not satisfactory. The object of testing in such cases is twofold: first, to select candidates who will be worth training, and, second, to insure that there will be no rejection of those who may be capable of performing flying duties. It is necessary not only that the candidate hear speech or signals; he must be able to interpret the message, and this involves not only hearing acuity but intelligence. If there is gross hearing loss in one ear only, the subject may be as competent to recognize signals as a normal person, with no hearing loss. In the case of flying personnel the question is further complicated by the noise level at which the work will be conducted. Consequently, tests with pure tones will not tell the whole story. A candidate's ability to understand from context must be determined by the use of sentences, which is a lengthy business. In an effort to simulate flying conditions, and at the same time to measure quickly a subject's ability to understand messages, gramophone records are used on which test words or sentences are spoken against a noise background. In this manner the acoustic conditions for testing can be reasonably well standardized, and, further, a number of candidates, using flying helmets with ear phones, can be examined at the same time.

LeJeune and Bayon, New Orleans.

Use of Sulfonamide Compounds in Otorhinology. Luis Vaquero, Prensa méd. mex. **6**:143 (Oct. 15) 1941.

According to the observations and careful statistics of Bilchick and O'Kane, the number of surgical interventions for acute suppurative otitis media in children is not reduced by chemotherapy. If this treatment is insisted on and no attention is paid to the operative indications, the disease is retarded but does not improve. On the other hand, the purulent discharge diminishes notably, its duration being reduced 50 per cent as compared with the control. In some cases the improvement is surprising, as suppurations which have lasted fifteen to twenty-five days may cease in twenty-four to forty-eight hours. If the operation has been insufficient and pus reappears beneath the mastoid scar, chemotherapy does not benefit and reintervention is indispensable.

Some authors think that sulfanilamide or one of its derivatives should be given routinely in every case of otitis media, but surgical operation on the membrana tympanica and on the mastoid may be used when indicated. Excessive confidence in chemotherapy delays surgical treatment and may lead to disaster.

Giffen, Toledo, Ohio [Am. J. Dis. Child.].

Indications for Operation on the Ear in Infancy. H. Flesch, Ann. pædiat. **156**:348, 1941.

There is no longer any doubt that occult mastoiditis occurs more frequently in early infancy than was formerly supposed. When protracted, irregular fluctuations of temperature have been noted in infants with otitis media and the child has lost weight in spite of careful nutrition, blood transfusions, etc., Flesch considers antrotomy to be indicated, after an adequate period of waiting, provided no symptoms of other complications have been observed. A precise term for delay cannot be fixed, as this depends on such factors as the amount of weight lost and the general clinical picture. The author's treatment was successful in most cases. When

inflammation of the middle ear is complicated by pneumonia, the latter disease should be cured first. Only in exceptional cases is it advisable to operate before. If the ear is involved in cases of toxicosis, paracentesis should be performed. Antrotomy should be resorted to only after every other treatment has failed. If one cannot find any alteration in the middle ear, the patient must not be operated on.

In cases in the author's experience in which the condition would otherwise have proved fatal cures have certainly been attained by antrotomy. But whether the operation appreciably reduces the high mortality of grave toxicosis is of course questionable.

AUTHOR'S SUMMARY [AM. J. DIS. CHILD.].

EXPLOSIVE TRAUMA OF THE EARS. RUEDI LUZIUS and BRUNO MÄDER, Pract. oto-rhino-laryng. **4**:177, 1942.

The authors had an opportunity to study 8 patients with ruptured ear drums as a result of an explosion in a munitions factory. Observations on these patients were made immediately after the explosion and continued for one and a half years. Marked loss of hearing for both conversational and whispered speech, with a typical picture of mixed deafness, was noted in all cases immediately after the accident. However, the final results varied considerably. In the cases in which there was early elimination of the infection in the middle ear and the perforation in the drum membrane closed over, with or without a small scar, normal hearing was restored. On the other hand, when the secondary infection of the middle ear became chronic and the tympanic perforation persisted, residual loss of hearing was noted, usually in the higher tone range.

The authors conclude that in spite of severe damage to the inner ear regeneration usually occurs and is only moderately influenced by any secondary infection of the middle ear.

PERSKY, Philadelphia.

THE VEGETATIVE NERVE SUPPLY TO THE INTERNAL EAR. W. RACINE, Pract. oto-rhino-laryng. **4**:205, 1942.

Racine repeated the experiments of Gaskell showing that the inner ear receives a nerve supply from the vegetative nervous system by two pathways—the periarterial route and the nerve trunks. The vegetative system terminates as a syncytium, which takes the form of either the "terminal reticulum" of Stöhr or the "vegetative group plexus" of Boeke. It is as the "terminal reticulum" that the author has discovered, both in the macula and in the crista, the parasympathetic nervous system, independent of the periarterial route.

PERSKY, Philadelphia.

INFLAMMATIONS OF THE TIP OF THE PETROUS BONE. HANS BRUNNER, Pract. oto-rhino-laryng. **4**:211, 1942.

Brunner reports a group of 15 cases of inflammatory disease of the petrous tip. In 3 of the cases the symptoms were not typical. Trigeminal neuralgia was prominent in 10 cases. This pain was centered in the temple in 6 cases, in the teeth in 4 cases and in the orbit in 3 cases. Hypesthesia (including loss of corneal reflex) was present in 5 cases and palsy of the abducens nerve in 6 cases. In reviewing these symptoms, the author states that as a rule if an inflammatory process in the petrous tip produces palsy of the abducens nerve, it also produces symptoms referable to the trigeminus, but if the inflammation produces trigeminal symptoms, it does not necessarily produce abducens palsy. There may be exceptions to this rule.

The symptoms referable to the petrous tip had their onset at different intervals. In 5 instances the symptoms appeared prior to any operation and in 7 cases after an operation (in 4 cases after a simple and in 3 cases after a radical mastoidectomy). In the first group the symptoms appeared at varying intervals after the onset of acute otitis media, i. e., from immediately to ten weeks; after a simple mastoidectomy they appeared in from five to thirty days, and after a radical operation the periods were two days, thirty-five days and seven years.

The treatment advocated by Brunner is either an extensive simple or a conservative radical mastoidectomy. In addition, he makes an extensive exposure of the dura of the middle fossa. This exposure of the dura does not necessarily have to extend to the petrous tip. His mortality rate in these 15 cases was 17 per cent.

PERSKY, Philadelphia.

Larynx

TRACHEOTOMY: A STUDY OF ONE HUNDRED CONSECUTIVE CASES. G. S. FITZ-HUGH, South. M. J. **34**:1116 (Nov.) 1941.

Fitz-Hugh reports a series of 100 consecutive cases of tracheotomy performed over a period of approximately ten years. In the series 56 patients were males and 44 females. Forty of the patients were 2 years of age or younger. The cases were classified according to the degree of respiratory difficulty which necessitated tracheotomy. In 35 cases there was no respiratory difficulty, but tracheotomy was performed electively, in anticipation of such a condition or to facilitate future therapy. In 46 cases respiratory difficulty was present and was often progressive. In 19 cases the respiratory difficulty was extreme, and tracheotomy was done as an emergency.

Of the pathologic conditions which required tracheotomy, a foreign body was responsible in 26 cases, being present in the air passages in 24 cases and in the esophagus in 2 cases. Diphtheria was the cause in 17 cases, carcinoma of the larynx in 12 cases, laryngotracheobronchitis in 10 cases, infection of the neck in 5 cases, trauma of the larynx in 3 cases, tuberculosis of the larynx in 3 cases, benign tumor of the larynx in 3 cases, abscess of the larynx in 2 cases and miscellaneous conditions in 19 cases.

In performance of a tracheotomy local anesthesia is favored in most cases. The vertical incision is the one of choice. Suturing of the wound is not favored. Local instillation of a solution of cocaine into the trachea is of value in the control of cough. In the series pneumothorax occurred as a complication in 3 cases. Postoperative hemorrhage occurred in 2 cases and was fatal in both. In 2 cases fatal bronchopneumonia followed an elective tracheotomy.

SINGLETON, Dallas, Texas.

THE RÔLE OF THE EPIGLOTTIS IN ANESTHETIC DEATHS. G. H. CAIGER, J. Laryng. & Otol. **57**:250 (May) 1942.

The object of this paper is to prove that the epiglottis is capable of playing a dangerous and unsuspected role during anesthesia. Some years ago the author encountered a case of respiratory arrest which he relieved by digitally disengaging an impacted epiglottis. Subsequently, he found "at least half a dozen" similar cases, and it is his conviction that the condition is not as rare as one might suppose. Four reasons, at least one of which is obscure, are given for the nonrecognition of impaction of the epiglottis: (1) a letter, of unknown content, in the correspondence columns of the *British Medical Journal;* (2) the prevailing anatomic and physiologic teaching that the epiglottis is an organ of little importance and not likely to cause trouble, in which connection Negus' and Starling's well known theories are discussed; (3) the increased use of endotracheal anesthesia, rendering impaction virtually impossible, and (4) lack of postmortem evidence. On two occasions the author attempted to prove by postmortem examination that epiglottic impaction had been the cause of death, but his facilities for satisfactory illumination were poor and, while a colleague "thought he saw something in the larynx," no substantial evidence was adduced to support the suspicion.

Briefly, it is postulated that muscular relaxation during anesthesia produces a backward tilting of the epiglottis, and the respiratory air column, instead of striking the posterior surface of the epiglottis, impinges on the anterior surface, forcing the organ still farther backward, so that it overlies and obstructs the larynx. Such a state can be recognized and relieved by digital manipulation. It is argued that such an occurrence may be frequent, but the usual methods of resuscitation, such as swabbing the throat, traction on the tongue and artificial respiration, are sufficient to disengage the epiglottis; hence the true nature of the trouble is never suspected. In the event of firm impaction of the epiglottis it is recommended that the tongue be drawn well forward with tongue forceps and the epiglottis disengaged by lifting it from the side with the finger.

LEJEUNE AND BAYON, New Orleans.

TRACHEOCELE: REPORT OF A CASE. GRENET, GAUTHERON and SAULNIER, Bull. Soc. pédiat. de Paris **37**:507, 1940.

The authors report a case of tracheocele in a boy of 3½ years, first seen five months before, when he was brought for relief of a soft tumor occupying the subclavicular spaces. This swelling appeared suddenly in the course of a violent fit of coughing, as noted by the mother. Examination showed a tumor as large as a medium-sized orange, soft, painless and sonorous on percussion. There was no crepitation, and its volume was augmented by effort; reduction could not be effected.

Aside from this, the child was entirely well. Several punctures were made, and air was withdrawn on each occasion together with a small quantity of a clear fluid, in which a few red cells were seen. The fluid was sterile, and laryngoscopic and roentgen examinations revealed no abnormalities. Injection of the tumor with iodized poppyseed oil showed that the cavity extended from the tracheal region a little below the larynx, but it was not possible to demonstrate a communication. This condition must be differentiated from pneumocele, which is crepitant and is always accompanied by functional disturbances, and from subcutaneous emphysema, in which crepitation is also found and in which the causative agent is usually readily apparent.

The tracheocele under discussion was doubtless due to congenital weakness of certain muscle fibers.

AMESSE, Denver [AM. J. DIS. CHILD.].

Nose

CONGENITAL CHOANAL OCCLUSION. WILLIAM L. BONHAM, South. M. J. **35**:252 (March) 1942.

The author briefly reviews the literature on congenital choanal occlusion and reports in some detail 2 cases of this rather unusual condition. He points out that Anderson, in 1937, stated that only 160 cases had been reported since 1830. The occlusion may be unilateral or bilateral, complete or partial and membranous or bony or both.

The chief symptoms are: (1) respiratory difficulty, which depends on the extent of the occlusion; (2) accumulation of thick, glairy mucus on the floor of the nose, which can be removed by the patient only by his leaning the head forward or by irrigation; (3) impairment of the sense of smell in cases of unilateral occlusion and loss of the sense when the condition is bilateral, and (4) great difficulty in nursing and breathing in the newborn. The diagnosis is made by the use of a probe, catheter or postnasal mirror and by digital examination of the nasopharynx. Roentgen examination is of value after instillation of iodized oil into the nasal cavities. The treatment is surgical and varies with the age of the patient. In older patients a submucous resection is performed, and the posterior bony and membranous portions of the septum are removed. The operation is completed by removal of the choanal obstruction. In infants and children the submucous resection is not done.

SINGLETON, Dallas, Texas.

SOME WHYS AND WHEREFORES RELATIVE TO NASAL SINUS HISTOPATHOLOGY. JOSEPH D. HEITGER, South. M. J. **35**:359 (April) 1942.

The author calls attention to, and condemns, the fact that from the era of Hippocrates to the present panaceas and nostrums have been foisted on a gullible public suffering from so-called catarrh and sinus disease. Inadequately informed physicians, medical journals, the radio and the magazine digests are credited with disseminating false propaganda on the subject of sinus disease. The author stresses the importance of accurate diagnosis and logical reasoning in the management of infections of the sinuses. He points out that allergy and vascular phenomena often complicate the picture and require differentiation in one's diagnosis and therapeutic efforts.

The histology of the mucous membrane of the sinuses and the histopathology of several types of diseased membranes are discussed in considerable detail.

SINGLETON, Dallas, Texas.

NASAL SINUS SURGERY: A CRITICAL REVIEW OF THE CAUSES OF UNSUCCESSFUL END RESULTS. A. R. HOLLENDER, South. M. J. **35**:363 (April) 1942.

According to the author, the assumption that the end results of operation on the nose and sinuses are highly satisfactory is refuted by data obtained from 154 rhinologists by the questionnaire method. The average proportion of "cures" resulting from surgical procedures on the nose is reported as 71 per cent and that from operations on the sinuses as 42 per cent. Hollander believes that the causes of the poor end results of rhinologic operations include: (1) inaccurate diagnosis; (2) lack of consideration of allergic factors; (3) failure to appreciate the influence of organic disease; (4) hasty surgical intervention; (5) incomplete and ill performed operations and (6) inadequate postoperative management.

For better results in the surgical management of sinus disease the author stresses the importance of a more accurate diagnosis, greater care in selection of patients for operation, greater consideration of the allergic aspect, more rational application of surgical procedures and better postoperative management.

SINGLETON, Dallas, Texas.

THE TREATMENT OF ATROPHIC RHINITIS BY ESTROGENIC COMPOUNDS. I. SIMPSON HALL and I. A. M. MCLEOD, J. Laryng. & Otol. **57**:337 (July) 1942.

A new treatment for atrophic rhinitis was inaugurated when Mortimer and his co-workers focused attention on the use of estrogen for inducing changes in the nasal mucosa. Hall and McLeod used three preparations, each made up in oil. They concluded that the synthetic estrogen was the one most suitable for their work. The method advocated for treatment was nasal douching, followed by the spraying of the estrogen preparation into each nostril at least three times a week for a variable period. No ill effects were noted during or after treatment, even when the therapy was employed for periods as long as twenty weeks. From a clinical standpoint the results of treatment are considered by the authors superior to those obtained by any other method.

LEJEUNE AND BAYON, New Orleans.

Miscellaneous

THE EFFECT OF BENZEDRINE SULFATE IN MIGRAINE. J. S. GOTTLIEB, Am. J. M. Sc. **204**:553 (Oct.) 1942.

The author states that dilatation and distention of cranial arteries, which may be produced by a variety of factors, form the basis of migraine headache. Ergotamine tartrate in adequate dosage is effective in many cases in aborting, reducing or terminating an attack of migraine headache because this drug decreases the amplitude of the pulsation of the cranial arteries, chiefly certain branches of the external carotid artery. Because amphetamine sulfate is a vasoconstrictor with prolonged action which also decreases the amplitude of the pulsations of the cranial arteries, its use in the treatment of migraine seemed logical. This drug was administered orally to some patients and intravenously to others in doses of from 3 to 20 mg.

Twenty-five patients suffering from migraine were treated symptomatically with amphetamine sulfate. Twelve of 18 patients (67 per cent) had their attacks aborted quickly when the drug was administered intravenously. Eight of 22 patients (36 per cent) obtained relief or had their paroxysms aborted when the drug was taken orally during the prodromal stage. Three patients having frequent and severe attacks became symptom free when the drug was taken daily and in divided doses.

DAVISON, Danville, Pa.

EFFECT OF FOOD AND ALKALI ON THE ABSORPTION AND EXCRETION OF SULFONAMIDE DRUGS AFTER ORAL AND DUODENAL ADMINISTRATION. O. L. PETERSON and M. FINLAND, Am. J. M. Sc. **204**:581 (Oct.) 1942.

The authors studied the results of administration of single 5 Gm. doses of the sulfonamide compounds to healthy ambulatory males and drew the following conclusions: 1. Sulfanilamide is absorbed rapidly and completely when it is given orally or injected into the duodenum. Food delays absorption of the drug but does not diminish the amount excreted in the urine. 2. Sulfapyridine, sulfathiazole and sulfadiazine are poorly absorbed from the duodenum. 3. Sulfadiazine is absorbed more slowly but more completely when it is given after a meal than when it is given on a fasting stomach. Alkali appears to increase the amount of drug absorbed when it is given with sulfadiazine after a meal. Sodium bicarbonate hastens the absorption of sulfadiazine when it is given with the drug on an empty stomach but does not increase the amount absorbed. 4. Sodium sulfadiazine is rapidly absorbed from the duodenum or after oral administration in a fasting subject. Absorption is delayed and is less complete if it is given after a meal.

DAVISON, Danville, Pa.

THE INFLUENCE OF THE FORMATIVE DENTAL ORGANS ON THE GROWTH OF THE BONES OF THE FACE. LAWRENCE W. BAKER, Am. J. Orthodontics **27**:489 (Sept.) 1941.

According to Baker, growth energy is stored within the formative organs of the teeth, and this energy is responsible for the rapid growth of the facial bones during childhood and adolescence. Experiments conducted on rats, cats and pigs by the removal of some of the formative organs (rats, incisors; cats and pigs, permanent teeth) disclose that there is an arrest in the growth of the facial bones on the side of the operation and in the direction in which the teeth experimented on would grow. The author regards forming teeth in animals and human beings, both temporary and permanent, as highly specialized epiphyses which act against the pressure of the growing bones of the skull.

LEWIS, Dayton, Ohio.

THE FUNCTIONAL PATTERN OF THE AUTONOMIC NERVOUS SYSTEM. DAVID HIGBEE, Ann. Otol., Rhin. & Laryng. **50**:1047 (Dec.) 1942.

Higbee starts out by stating: 1. The autonomic system is that part of the nervous system which presides over involuntary functions. 2. It is divided into two portions. One, known as the sympathetic, regulates adjustments to external environment. The other, called the parasympathetic, presides over the functions which are vital to existence. Together their activities embrace the whole fabric of metabolism. 3. This system is a motor or efferent system. It does not carry sensations of pain. Its fibers are distributed to gland cells and to the cardiac muscle and the smooth muscle of the viscera and the blood vessels.

He reviews the embryology, anatomy and physiology of the autonomic system. The role of the sympathetic system is that of an emergency mechanism. It does not act constantly and is not essential to life. It is stimulated through the special senses for the most part, in adjusting man to his external environment. It is also stimulated by adverse conditions within the body, such as pain, hemorrhage, dehydration, infection and emotional states. When it is stimulated, the hair is erected, the pupils are enlarged, the heart rate is increased, the blood pressure is elevated, the great splanchnic vessels are contracted, the peripheral circulation is increased and the sphincters are contracted. This stimulation is accomplished by the discharge of epinephrine into the blood and the activation of sympathin at the myoneural junction. The parasympathetic system functions constantly, and its role is to maintain a state of health within the body. It provides for the storing of energy and for reparative processes. It effects digestion and elimination. It is concerned with immunity and sensitivity, and it controls the activities of most of the endocrine glands. When activated, it contracts the pupil, slows the heart, increases the tonus of the gastrointestinal canal and empties the hollow viscera.

The law of isochronism supports the theory that sensations of pain are not conducted over autonomic fibers. This law is that between a muscle and its nerve there must be similar reaction time inherent in the two tissues independently of each other. It has been shown that whenever these reaction times differ more than 100 per cent the impulse is blocked. It has been found that heavily myelinated fibers of the sensorimotor tracts conduct stimuli at the rate of about 100 meters a second. The fibers of the autonomic system, which have little or no myelin, will conduct stimuli at about 12 meters per second.

The autonomic system must be regarded as a part of the whole nervous system and is neither wholly dependent on the latter nor totally independent of it. Psychic stimulation certainly involves the autonomic system, although the cortical areas transmitting the influence have not yet been determined. Experiments show an inhibitory influence of the cortex on the hypothalamus, and there is strong evidence to show that control centers for the sympathetic and parasympathetic systems are located in the latter.

The sympathetic and parasympathetic systems are antagonistic and maintain a balance which swings one way or the other according to the needs of the individual. Normally the parasympathetic nerves maintain the function of vital organs, but under stress the sympathetic nerves become dominant.

Parasympathetic fibers are stimulated by insulin, choline, acetylcholine, pilocarpine and eserine and paralyzed by alkaloids of the atropine group and by nicotine. Sympathetic fibers are stimulated by the adrenal medulla and the thyroid and by ephedrine and benzedrine. The motor and sensory fibers are paralyzed by ergonovine and ergotamine, but not the inhibitory fibers. Nicotine paralyzes all fibers of the autonomic system. Experiments indicate that acetylcholine is the mediator between the nerve endings and the end organ in the parasympathetic system and sympathin in the sympathetic system.

When pain is a symptom, it is due to somatic or visceral sensory fibers, as no afferent neurons have ever been found. By a multiple distribution of sensory fibers to both voluntary and involuntary structures a diffuse response may result from a stimulus perceived through a single sense organ.

M. V. MILLER, Philadelphia.

Society Transactions

CHICAGO OPHTHALMOLOGICAL SOCIETY AND CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

LOUIS G. HOFFMAN, M.D., *President, Chicago Ophthalmological Society*

AND

G. HENRY MUNDT, M.D., *President, Chicago Laryngological and Otological Society*

Joint Meeting, Feb. 8, 1943

SYMPOSIUM ON PAIN IN THE HEAD AND FACE

Headache as a Problem in Internal Medicine. DR. G. W. SCUPHAM.

The lay public has been so well educated to the fact that eyestrain and diseases of the eye are frequent causes of headache that it will go to the ophthalmologist; the otologist, too, has his turn because of the belief that "sinus headache" is common. The problem of headache is a difficult one because of the multiplicity of conditions of which it is the outstanding symptom. From the standpoint of the internist, not only the diagnosis is difficult but the therapeutic problem is often more so. If he limits himself to any one of the fields of medicine in which headache is important, he is likely to fail to arrive at a conclusion in a given case.

A number of mechanisms may be involved in the various types of headache, but from the standpoint of pain in the head and face these are not so important. Pain may be the result of local disease or local reflex phenomena. Some vascular disorders are characterized by pain. Arteritis of the temporal vessels results in a throbbing, burning pain localized in the region of these vessels, often associated with evidence of an inflammatory process. Herpes zoster is a fairly common cause of pain in the head. Tumors of the scalp and skull, multiple myeloma and osteitis of the skull may also be mentioned.

Headache is more important to the internist than pain in the head or face. The character of the pain seems to be different. Headache arises within the head, whereas pains in the head and face are more or less superficial. Headache varies greatly with different persons. It is uncommon in children, and in aged people it is less common than in persons of middle life.

The majority of headaches are merely part of the general symptoms of the disease. Headache is an important symptom of almost all acute infections. Typhoid is often characterized by long-continued headache, and influenza and acute infections of the respiratory tract have an onset with headache. A kind of toxic headache is that resulting from sulfathiazole; alcohol intoxication, as well as carbon monoxide poisoning, also causes toxic headache, but the latter often results in structural changes. In acute nephritis headache, associated with nausea, is an important symptom at the onset. The headache is in part toxic, but a rise in intracranial tension may also be a factor; in the uremic phase of chronic nephritis the headache is probably of toxic type. Headache is a common, and extremely important, symptom of chronic malaria; it is persistent and severe and disappears with control of the disease.

Hypertension is the major vascular disease of which headache is an important symptom. Inhalation of amyl nitrite results in headache and fall of blood pressure. This presents an interesting contrast in the mechanism of headache, as in persons with hypertension a fall in blood pressure often causes the headache to disappear. Many persons with severe hypertension never have headache, and many with moderate hypertension complain of severe throbbing headache, often continuing for days, without notable exacerbation of the blood pressure. In general, patients whose hypertension is in the malignant stage are subject to severe headache, probably because there is pronounced vasoconstriction of the arterioles of the central nervous system.

The headache which resembles the type caused by injection of histamine may be confused with migraine because it is often unilateral. With this type of headache there is no history of familial headache; the headache often begins at night; there are often flushing of the head and face on that side and tenderness over the temple and the carotid artery. Pressure on the carotid artery may result in relief. A small amount of histamine injected subcutaneously may initiate an attack. The recognition of this type of headache is important because relief may be obtained by the therapeutic administration of histamine.

There are a great variety of headaches associated with rheumatic conditions, from the headache of acute rheumatic fever, which may be mistaken for that occurring with tumor of the brain, to a mild occipital headache of chronic nature. Perhaps the most common type is that associated with fibrositis and myositis. Another frequent type occurs with cervical osteoarthritis and is associated with increased tension of the muscles of the spine and neck.

Reflex headache is more difficult to differentiate. It is often associated with disease of the pelvic structures. Constipation is said to cause headache reflexly, and evacuation of the lower portion of the bowel brings relief.

The typical features of migraine headache are well known. A family history of the disorder is so common that if it is not obtained the diagnosis is doubtful. The headache develops slowly, with nausea and vomiting, followed by relief and sleep. The headache is often preceded by aura. The disease is extremely variable in some persons, and it sometimes disappears at or near the menopause. Another striking feature is its association with the menstrual cycle. Migraine may be precipitated by a wide variety of factors: food allergy; emotional states; certain endocrine abnormalities, particularly the gonadotropic factor; fatigue and infection. It is commonly thought to be incurable, but much can be done if the particular patient is studied carefully. Treatment should be directed toward elimination of the exciting factors when possible.

Neurologic Aspects of Headache. DR. LEWIS J. POLLOCK.

When a stimulus is applied to the skin or mucous membranes, sensory impulses are transmitted by the sensory fibers of the craniospinal nerves and reach consciousness directly through their appropriate sensory tracts. Thus, inflammatory and other pathologic processes of the skin and mucous membranes produce impulses which are carried by ordinary sensory cutaneous nerves and are localized to the area stimulated.

When the subcutaneous areas are stimulated, sensory impulses are transmitted in two ways: first, by the ordinary sensory components of craniospinal nerves carrying deep sensibility—in the case of the head and face, by way of the sensory components of the third, fourth, fifth, sixth, seventh, ninth, tenth, eleventh and twelfth cranial nerves—and, second, by a more complicated mechanism producing referred pain—for example, the pain in the shoulder tip which occurs when the central portion of the diaphragm is stimulated by diaphragmatic pleurisy. Here, the impulse travels by way of the phrenic nerve through the posterior roots of the fourth and fifth cervical nerves to the spinal cord, there synapsing with the descending short fiber tracts to the points of origin of the cervical sympathetic fibers, and then through the preganglionic and postganglionic fibers to the skin, where a chemical change is produced which stimulates the sensory end organs. Sensory impulses are then carried by the sensory spinal nerve into the spinal cord and by its appropriate sensory tract into consciousness. The pain is thus referred to the skin over the shoulder tip.

The same mechanism applies to pain in the head and face. In the case of vascular headache, and perhaps migraine, stimuli occur as the result of dilatation of the blood vessels; impulses travel by way of the nervi vasculorum, originating in the cranial nerves, and perhaps also in the upper cervical nerves, and find their way into the brain stem and the medulla; there a synapse with the short descending pathway of the cervical sympathetic chain occurs; impulses are sent over the postganglionic fibers to the skin, where, again, chemical changes produce a stimulus in that cutaneous area and impulses carried by the particular sensory nerve involved, chiefly the fifth, enter the brain stem. This holds true also for distention of other subcutaneous tissue, as well as for traction on the meninges and the dural sinuses. This pathway can be proved by experiments in which the pain resulting from faradic stimulation of the superior cervical ganglion can be abolished by section of the sensory root of the fifth nerve. The pain cannot be cured by severing the preganglionic fibers when the appropriate anterior roots are cut or by sectioning the cervical portion of the sympathetic chain below the superior cervical ganglion.

Usually pain in the head is not the principal symptom of intracranial disease; although headache may be the presenting symptom of intracranial tumor, it is not the only one. Diplopia, hemiparesis, facial paresis, ataxia, loss of visual acuity, vomiting and mental disturbances are at times associated with the headache. Headache, when present, is associated with vomiting, which may be projectile and usually does not lead to alleviation of pain. Here the headache is sharp, severe and continuous; it may be a throbbing, stabbing, boring pain. It is often brought on by sudden change of posture and may disappear on change in the position of the head; in cases of intraventricular and infratentorial tumor, the patient may hold his head in a position which tends to lessen the possibility of occurrence of pain. The pain may be asso-

ciated with suboccipital tenderness and rigidity of the neck. It is increased with jolting and straining, and with sudden exacerbation of pain vomiting may occur.

The location of the pain is not characteristic, nor is it an index to localization of the tumor. The character of the tumor may influence the type of headache and its severity. A rapidly growing tumor is associated with more headache; a hard tumor causes more pain; a circumscribed tumor produces pain of greater intensity than does an infiltrating tumor. Percussion of the skull at times serves to demonstrate an area of circumscribed tenderness, which may indicate intracranial disease.

Associated signs and symptoms serve as clues to the diagnosis. Hemiplegia of sudden onset points to hemorrhage, thrombosis and embolism; the sudden onset of excruciating suboccipital pain with loss of consciousness, in rare instances hemiplegia and rigidity of the neck characterizes subarachnoid hemorrhage, which is usually due to a ruptured cerebral aneurysm. Inflammation of the leptomeninges alone or with encephalitis is associated with rigidity of the neck or Kernig's or Brudzinski's sign, and evidence of inflammatory disease is encountered on examination of the cerebrospinal fluid. Serous meningitis with paroxysmal hydrocephalus is characterized by attacks of headache, vomiting, sometimes choked disks and signs of interference with cerebral function. Such attacks, after a relatively short course, may disappear and a considerable time elapse before another occurs. In cases of this type one may often obtain a history of another anaphylactic phenomenon, such as angioneurotic edema or Ménière's syndrome.

Most cerebral diseases produce interference with motion, sensation, tone, synergy, speech, reflexes and functions of the cranial nerves. Only a consistent decrease in the activity of the deep reflexes on one side may be noted, but when this is combined with diminished superficial reflexes on the same side, organic disease is strongly indicated. Changes in the visual fields may be noted long before a change in the optic nerve is evident, especially with tumor of the temporal lobe of which quadrantic anopsia and headache may be the sole symptoms. Whether encephalitis uncomplicated by meningitis may produce headache cannot be stated. Many of the encephalitides are associated with meningitis, and patients with lead encephalopathy may exhibit a notable change in renal function, which in itself may produce severe headache.

In a large number of patients suffering from headache careful search may reveal no organic disease. Such headache may be termed "psychogenic," or, perhaps better, headache associated with neurosis. Usually when the patient complains of headache, he does not mean that he has pain in the head. The feeling is sometimes described and at times not; always it is described as "intense," "terrible," "horrible," "splitting" or "unbearable." It is the hyperbole and exaggeration which point to the condition. The sensations complained of are also myriad. The so-called pain may involve any part of the head and may shift from day to day or from hour to hour. It is continuous and is usually unaffected by weather or by ordinary changes in position. It is not accompanied by nausea or vomiting. It is always increased by the making of decisions and by work. It is relieved by reassurance, whether by a physician's statement or by favorable observations by the patient. If the patient becomes interested in some pursuit, the severity of the headache will abate as long as the interest lasts; it is decreased by any new treatment or doctrine. It is worse in crowds and is increased by excitement, disputes, quarrels and domestic, social and business conflicts.

As with sensations associated with phobias relating to the heart or stomach, so with this headache. A real pain or discomfort of organic origin may often have preceded it; it may follow an attack of migraine or an indurative, an ocular or a sinus headache, or it may coexist with any of them. Prominent among the symptoms are depression, abulia, inattention, difficulty in concentration, forgetfulness, introspection, anxiety, fears, indecision, fatigue and insomnia. Not only is it important to distinguish "psychogenic" headache from headache of organic origin, but it must be remembered that the attacks may be the beginning of a more serious condition, such as the onset of melancholia, dementia praecox or dementia paralytica.

Often, pain in the head or certain types of headache are classified as neuralgia, such as auriculotemporal and occipital neuralgia. This, I believe, is a mistake. If one accepts trigeminal neuralgia as typical of the neuralgias, then sudden paroxysms of severe pain in the distribution of the sensory supply of the nerve, lasting only a fraction of a second, and brought about by the touching of a trigger or an algogenic zone, are seen only in relation to the trigeminal or the glossopharyngeal nerve.

The characteristic neuralgia affecting the trigeminal nerve is observed usually when no underlying pathologic process can be demonstrated, even on inspection of the gasserian ganglion. At times it occurs with tumor of the gasserian ganglion rarely with tumor of the cerebellopontile angle and sometimes with multiple sclerosis. It is not related to any other extraneural condition, such as disease of the sinuses, teeth or other structures of the head and face. The characteristics of the pain are unique. It is always referred to the same zone of distribution

—the supraorbital, the infraorbital or the mental or to two or all of the branches of the nerve. It may be seemingly spontaneous, but can always be brought on by touching the trigger zone or by movements stimulating such a zone, such as those of talking, chewing and swallowing. The area stimulated is not always the area in which the pain occurs. The pain is severe, sharp, shooting and electric-like, lasts but a few seconds or may consist of a succession of shorter pains. There is no relief other than interruption of the sensory fibers of the fifth nerve.

Allergic Aspects of Headache. DR. SAMUEL M. FEINBERG.

From the allergic standpoint there are two main types of headache: the classic migraine and the simple, but frequently recurring, headache. I believe there is no real etiologic distinction between the two types; the migrainous headache is merely more severe. I have observed many patients whose ordinary, recurrent headaches develop into migraine, and other patients whose headaches vary at different times from the simple form to the typical migraine headache.

What is the mechanism of migraine? It has been demonstrated that angioneurotic edema produces cerebral symptoms and that cerebral vessels have a vasomotor control. Several years ago a young woman who had violent migrainous symptoms was operated on for suspected tumor of the brain. Severe edema of the brain was noted, but no tumor was observed. A defect was left in the skull after operation, and it was then observed that the onset of a migrainous attack was initiated by preliminary pallor of the face and depression of the area in the skull. As the attack progressed, the area of decompression bulged. Allergic studies showed clinical sensitivity to specific foods, the latter precipitating the headaches and the bulging of the area of decompression. It appears, therefore, that the mechanism of migraine is probably a cerebral angiospasm, followed by vasodilatation and increased intracranial pressure.

What evidence is there for the allergic basis for headache? Dr. Scupham has already mentioned the hereditary tendency to migraine. The majority of patients with migraine give a family history of the disease, but a large percentage have other allergic disturbances in the family. It is generally overlooked that allergic patients exhibit a tendency not only to the inheritance of allergy but to the predominance of special allergic manifestations in the family. Offspring of persons with urticaria are more likely to have urticaria, and those of persons with migraine, to have migraine than any other allergic manifestation. That migrainous attacks are frequently precipitated by nonallergic factors, such as fatigue, constipation, excitement, changes in the weather and phenomena associated with the endocrine cycle, does not detract from the allergic concept of the disease. Such precipitating factors may act as the "trigger" to set off allergic phenomena, such as asthma, rhinitis and urticaria. It is true that only a small proportion of patients with migraine, perhaps a fourth, respond to cutaneous tests. Yet with some of the other allergic manifestations, such as urticaria and gastrointestinal allergy, the incidence of cutaneous reactions is also small. By clinical tests and by observation it can be determined, in spite of negative cutaneous reactions, that the headache of many patients is due to foods.

By means of cutaneous tests, simple observations, diary records and restricted diets a diagnosis can usually be made. It may be stated conservatively that half of such patients can be shown to be allergic; for the headache of the others we believe no satisfactory explanation exists at present. The majority of the allergic patients are sensitive to foods, the most common of which are wheat, milk, cocoa, peas and beans, potato, pork, egg and onion. It should not be forgotten that inhalant allergens may also cause migraine.

Endocrine Factors. DR. HUGO R. ROSY.

It is to be expected that endocrine glands play some etiologic role in headache, for three reasons: 1. Two glands, the hypophysis and the pineal body, are located in the intracranial cavity. 2. Hormones are known to affect secretion of the cerebrospinal fluid, as well as retention of water in various tissues, including possibly the meninges and brain. 3. Glands participate in a number of factors known to be concerned in the production of headache, such as control of blood pressure, certain metabolic processes, activity of the autonomic nervous system and the level of sugar, calcium, sodium and other constituents of the blood.

It is difficult to obtain reliable statistical data with regard to the frequency of endocrine headache, inasmuch as the diagnosis of endocrine disturbance is sometimes a matter of opinion. Further, even in the presence of definite endocrine disturbance, its role in the production of the headache in a given case may be open to question. It is probably safe to say, however, that the number of cephalalgias in which glandular disturbance is a contributory factor is high, while true, or primary, endocrine headache is relatively rare.

In cases of tumor or of hyperplasia of the hypophysis headache is a prominent symptom; it is more or less constant and is bitemporal or occipital. It is believed to be due to distention of or pressure on the capsule of the gland or the dural diaphragm above the gland. Eosinophil, basophil or chromophobe tumor may produce such headache, regardless of the functional state—hypoactivity or hyperactivity—of the gland. Removal of the tumor or high voltage roentgen therapy may completely relieve the headache. There is some evidence that the headache of pregnancy, the headache of the artificial or the natural menopause and the so-called premenstrual headache may be of pituitary origin, being caused probably by mechanical effects of the hyperplasia of the gland which is known to occur under these conditions. Such headache may be successfully treated by large doses of estrogen preparations, which are known to exert an inhibitory effect on hyperplasia of the pituitary.

Tumor of the pineal gland may produce severe headache, of the type seen with brain tumor, especially if the growth leads to compression of the sylvian duct and causes internal hydrocephalus, with consequent increase of intracranial pressure.

Headache is not infrequently associated with disturbances of the thyroid gland, either hyperthyroidism or hypothyroidism. Such headache is usually mild, with no characteristic localization. That it is related to the disturbance of the thyroid is shown by the relief afforded by specific therapy—administration of thyroid in cases of hypothyroidism or thyroidectomy in cases of hyperthyroidism.

Headache is a prominent symptom of Addison's disease or other forms of adrenal insufficiency and may be relieved by administration of adrenal cortex extract. In hyperinsulinism headache may be an element of the hypoglycemic syndrome. Characteristic of this headache is its onset when the stomach is empty and its prompt relief with intake of food. Hyperglycemia, on the other hand, does not in itself produce headache; however, headache frequently accompanies diabetic acidosis, and severe headache in a case of uncontrolled diabetes may be a danger signal of impending coma.

Headache in various conditions of doubtful glandular origin may present a considerable diagnostic and therapeutic problem. Thus, it may be difficult to decide whether headache in cases of a hypotension-fatigue syndrome, of certain ovarian disturbances or of the male climacterium is of glandular or psychic origin. Even therapeutic success with specific glandular preparations may not determine the nature of the headache, in view of the possible suggestive effect of treatment. With certain forms of obesity, especially when the condition is accompanied by hyperostosis frontalis interna (Morel's syndrome) or by painful lipomatosis (Dercum's disease), headache may be a prominent complaint. Reduction of weight in cases of such disturbances is usually followed by more or less complete relief of the headache, probably because of the lowering of the blood pressure and the lessening of water retention as a result of loss of weight.

Ophthalmologic Aspects. DR. BEULAH CUSHMAN.

Patients with headache come to the ophthalmologist sooner or later, as it has become almost axiomatic that errors of refraction and muscle balance cause headache and asthenopia, and this belief is fostered by advertisements. The eye is particularly sensitive to neurosis, since it is a highly specialized organ.

Of the twelve cranial nerves, four go directly to the eye and three have distributions to the eye. The extensive distribution of the fifth nerve from the midbrain down to the second cervical level and the numerous interrelations which it forms with the nuclei of origin of all the other cranial nerves constitute one reason that headache results from so many different causes and is so vague in distribution. The association of the trigeminus with the vagus nerve accounts for the vomiting which accompanies acute glaucoma. It also explains the relief of dyspepsia obtained by proper correction of errors of refraction and muscle balance.

Ocular headaches have been classified under three types: headache due to retinal irritation; headache due to organic disease and headache due to eyestrain.

Retinal irritation may follow glare produced by any sudden change from a dull to a bright light or by the use of a torch in industry. The range of sensitivity varies with the patient, depending in part on the amount of pigment in the pigment layer of the retina and the choroid. Ophthalmic migraine, a rare condition, may be precipitated by glare and has been described in association with palsy of an ocular nerve, generally the third; at first the palsy is temporary, later becoming permanent. The pain is diffuse, beginning in the temporal region and spreading to the occiput. Pain and nausea always precede the paralysis. The pain of ophthalmic migraine is usually on the same side as the hemianopia, whereas the pain of typical migraine is on the opposite side. The paralysis, which may involve the iris and the muscle of accommodation, usually passes off in the first attack. The attacks occur only a few times during the

patient's life, but each attack may continue for days, weeks or months. The involvement of the oculomotor nerve has been constant in all published cases, but the pathologic picture shows no uniformity.

Organic disease may produce ocular pain, which is dependent on the degree of abnormality of the ocular nerves and muscles. Sensation and discharge of the foreign body type are present, with inflammation of the conjunctiva, cornea and sclera.

Ciliary neuralgia is present with iritis and cyclitis, is usually worse at night and varies in intensity. Neuralgia of the eye has also been described as pain on movement of the eyeball and tenderness of the ocular muscles after exposure to cold air. Closely allied to this condition is neuralgia of the forehead. The symptoms may be associated with transient paresis, which responds to the use of heat and salicylates.

The pain of acute glaucoma extends along the distribution of the fifth nerve. With both chronic and secondary glaucoma a neuralgic type of pain is referred to the face, cheek and temporal areas and is often mistaken for the pain due to sinus infection until vision becomes affected.

Retrobulbar neuritis produces pain deep in the orbit and is increased on rotation of the eye; this pain may be present several days before vision becomes blurred.

Pain associated with proptosis, as seen with thyrotoxicosis, orbital tumor and cellulitis, varies according to the degree of distortion of the oculomotor muscles and nerves and the exposure of the cornea.

According to Butler, a refractive defect which has been present for many years is unlikely suddenly to exert a malign influence, unless some other factor, such as overstudy, work under unfavorable conditions, debilitating illness or psychic disturbance, is added. Headache of recent origin should have a recent cause.

Ophthalmic headache is presumably due to overwork of the muscles of accommodation and fixation; the pain is at its height late in the day and disappears after rest. The infrequency of headache in persons with monocular vision is said to be due to the absence of effort made for fusion through complex actions. Overaction of the ciliary muscle controlling accommodation, with production of fatigue and spasm, may be followed by headache. Refractive errors predispose to headache, as does muscle imbalance. The insertion of the frontalis muscle into the upper lid of each eye makes possible participation in the contraction of the orbicularis palpebrarum; the frontalis muscle on contraction pulls on the epicranial fascia, and this in turn pulls on the occipital muscles, with resulting fatigue and spasm.

Headache of ocular origin probably most often occurs in the presbyope with a receding near point. Many persons complain of ocular discomfort who have high degrees of hypermetropia and astigmatism or heterophoria which had caused no previous handicap. Aniseikonia is a cause of discomfort in patients with unrelieved symptoms of asthenopia or photophobia and may be suspected in the presence of anisometropia, in a patient with unequal accommodation, who wears lenses of unequal curve or thickness; in patients with space perception anomalies; in a patient with good vision in each eye who prefers to use one eye only, and occasionally in a patient with reading disabilities or migraine.

Otolaryngologic Aspects. DR. ARTHUR W. PROETZ, St. Louis.

Reliable statistics indicate that probably less than 5 per cent of headache is due to sinus trouble. Undue attention has been given to sinus headache by the public, which recently has been plied with semitechnical literature on sinusitis, while being kept in ignorance of the thirty other causes of headache. The physician must guard against acceptance of the patient's diagnosis merely because some sinus infection is present. Many persons have headache; many have infected sinuses, and the two conditions are often not related.

While the mechanism of headache is not known, much light has been thrown on it in recent years. It is usually associated with stretching or distortion of certain portions of the meninges or with the dilatation of certain arteries, notably the middle meningeal. The sinuses are closely related to these structures. Congestion and inflammation in the sinuses may produce typical headache.

However, such headache may not be referable to the sinuses themselves but may be of a vascular origin and result from conditions elsewhere in the body. It is my impression that some people are headache subjects, that is, that stimuli of various types produce vasomotor disturbances which set up the headache mechanism and produce the symptom characteristic of the person.

Mere stretching of the nasal mucosa does not produce pain. A number of cases have now been studied in which sudden edema stretched the lining of the sinus to ten times its normal

thickness without any subjective symptoms. It does not follow that stretching of the periosteum would be painless. This may be analogous to diffuse otitis externa, which is exceedingly painful.

The existence of a "vacuum" pain of any duration is open to doubt. I can find no evidence in the literature of any experimental proof that it exists. No physiologist whom I have consulted is willing to say that the sinus mucosa absorbs oxygen. It is pointed out that a system of tissues may react specifically in absorption or in secretion of a gas. If no selective action exists in a sinus, and only the usual exchange of gases occurs, then an amount of carbon dioxide must be given off by the membrane equivalent to that of the oxygen which it absorbs, and since carbon dioxide has a slightly greater volume than oxygen, a positive pressure must result if the ostium is closed.

Another possible mechanism, which I have not seen described, is the sudden engorgement or sudden collapse of a sinus membrane in the presence of a closed ostium, conceivably producing a positive or a negative pressure. Whether such pressures produce headache I do not know. In certain persons slight exhaustion of air from the nasal fossae does produce headache.

Observations on pain in cases of known acute conditions of the sinus—for example, acute empyema of the frontal and maxillary sinuses—lead to the conclusion that pressure exerted on an acutely inflamed area causes severe pain. When the pressure is relieved the character of the pain changes. Although the mucosa is still acutely sore, the pain in no way resembles a so-called sinus headache as it is understood. It is not gone in half an hour. One is led to conclude that a sinus headache which comes and goes is the result of vascular disturbance (inside or outside the sinus), and not of a fixed pathologic change.

Some nasal headaches can be demonstrated to result from the impact of jets of air during inspiration against the nasal ganglion or its branches. These jets are the result of constricted airways. The direction is determined by the topography of the nose and its irregularities.

It is difficult to obtain material for balanced judgment from the literature on headache. Many authors are themselves victims of the malady, and their accounts and interpretations are often highly tinged by their own experiences. This is entirely understandable, since a symptom so completely subjective can be best studied by its victims.

There remains the problem of the practical approach. Any one seeing many headache patients (and who does not?) must be struck by the fact that these persons are likely to make their own diagnosis, which leads them to the rhinologist or the ophthalmologist or the dentist or the allergist or, what is more likely, to each one in succession. Such a patient is commonly told: "It isn't your eyes; it must be your sinuses," or "It isn't your sinuses; it must be your teeth." He seldom is told: "Your headache is due to one or two or more of some thirty possible causes, and I shall try to find out which one it is."

In my own hands, the most direct means of arriving at some indication of the nature of a headache is a complete history, both of the headache from the standpoint of the attack and its background, and of the patient in general. A complete printed questionnaire helps greatly to visualize the case as a whole and to evaluate the data. Important, also, is a complete daily record of the patient's contacts, diet and activities. Again, a prepared chart with which I am thoroughly familiar has many advantages over a haphazard diary, or even a diary carefully recorded according to some system devised by the patient. Since the important points in such a diary are often the minute details, standardized records are of great help.

(Copies of the forms used were projected on the screen).

In the treatment of vascular headache, and, in fact, of some other forms, I find the oral administration of ephedrine with one of the barbiturates very useful in giving relief while the investigation is going on.

I may add that many headaches with the characteristics of sinus pain disappear on the administration of thyroid. A low basal metabolic rate is not always characteristic of this type of headache. I have commonly noted improvement with administration of thyroid even when the rate was +5 per cent or more.

DISCUSSION

DR. PETER BASSOE: It might have been well if some one had discussed recent research on the mechanism of headache. At the January 1943 meeting of the Chicago Neurological Society, Dr. Harold Wolff reported on the work done by his group in New York (Wolff, H. G.: Mechanisms of Headache, *Arch. Neurol. & Psychiat.* **50**:224 [Aug.] 1943). They made innumerable experiments on persons subjected to craniotomy and determined that the most sensitive areas are near blood vessels, an observation which is analogous to the situation with respect to the peripheral nerves. The median and sciatic nerves, with their arteries, surrounded by nerve fibers, are the areas most frequently affected by causalgia.

One speaker is missing on this program; no one discussed the role of the teeth. Even in this enlightened age a great many people have been deprived of teeth without good reason. A striking example is a woman with beautiful teeth who had pain which she localized in two teeth. These were removed and found to be normal, but the pain shifted to other teeth, which were also removed, until she had lost many teeth at the time she came under my observation. Questioning revealed that she was in a disagreeable situation from which she could see no escape. It was pointed out to her how this problem could be straightened out, and the pain disappeared and the rest of her teeth were saved.

Dr. Feinberg's comments on edema and allergic conditions as causes of pain are interesting. In 1 patient there developed a syndrome of cerebral tumor, with choked disk, vomiting and severe headache, but no localized signs. A decompression was made, and the patient recovered and had no further symptoms. No tumor was seen. He moved to another part of the country, and then typical angioneurotic edema developed. Undoubtedly, his symptoms of brain tumor were due to edema of the brain.

Another patient, a woman, had sudden severe headache referable to the brain, with aphasia and hemiplegia. She had been taking care of a child with scarlet fever, and abscess of the brain was suspected. She became comatose. The brain was explored, but only edema was seen. She recovered, but temporary aphasia recurred at each menstrual period.

Dr. G. W. Scupham: Dr. Feinberg's conception of migraine as essentially an allergic phenomenon is generally accepted. Migraine is analogous to asthma in many respects. That the factor which precipitates the episode is not always allergic does not alter the pattern. Just as asthma may be precipitated by many exciting factors, entirely foreign to allergic influence, so may migraine. The endocrine aspects deserves more consideration. It is difficult to say just what part the endocrines play in the mechanism of headache.

Dr. Proetz's scheme for the study of headache is splendid. That a patient has infected sinuses does not mean that this condition is the cause of the headache. The same holds true for hypertension.

Book Reviews

Primer of Allergy. By Warren T. Vaughan, M.D. Second Edition. Price $1.50. Pp. 176. St. Louis: C. V. Mosby Company, 1943.

In this pocket-sized volume Vaughan has made a lucid presentation of the general factors underlying the allergic state, as well as the individual problems of diagnosis and treatment as they confront the patient and his physician. The book is addressed to the lay reader, who should receive from it a good understanding of the allergic diathesis, its hereditary aspects and such specific concepts as allergens and shock tissues.

Such diagnostic measures as cutaneous tests, elimination diets and food diaries are impartially analyzed, and the lay reader is made aware of their shortcomings, as well as their advantages. He learns, too, that although there are definite therapeutic limitations in this field of medicine, at least 70 per cent of properly managed patients will obtain satisfactory relief of their symptoms.

In the reviewer's opinion, too much stress is placed on the role of food allergy, while too little attention is given to the problem of bacterial allergy. The latter is of considerable importance to a large number of patients suffering from asthma and allergic coryza who show completely negative reactions to cutaneous tests with the common inhalant and food allergens.

An excellent feature of the book is the inclusion of a question and answer section at the end of each of several of the chapters. These serve to explain in simple terms many of the concepts which might otherwise perplex the patient. The detailed instructions for the avoidance of the various allergens and the methods of keeping food and inhalant diaries are well formulated, and should be of considerable help to the allergic patient.

News and Comment

AMERICAN BOARD OF OTOLARYNGOLOGY

The next examination of the American Board of Otolaryngology will be held in Chicago, at the Palmer House and the Illinois Research Hospital, on Oct. 6, 7, 8 and 9, 1943. Candidates should apply at once to Dr. Dean M. Lierle, secretary of the board, University Hospital, Iowa City.